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SLEEPING SICKNESS.

UGANDA PROTECTORATE. **Annual Report of the Human Trypanosomiasis Research Institute for the Year ended 31st December, 1935** [DUKE (H. Lyndhurst), Director].—11 pp. 1936. Entebbe. Govt. Printer. [Shs. 1/50.]

This report covers the last year of the life of the Human Trypanosomiasis Institute at Entebbe.

The year was spent in an attempt to complete certain lines of research, *viz.*, the conclusion of the investigation of the efficacy of Bayer 205 as a prophylactic against the trypanosomes of man; and a continuation of the study of antelope as a reservoir of these trypanosomes. All this work has already been published, and has received notice in this *Bulletin*. *W. Yorke.*

CONGO BELGE: RAPPORT SUR L'HYGIÈNE PUBLIQUE AU CONGO BELGE PENDANT L'ANNÉE 1934 [VAN HOOF (L.)]. [Trypanosomiase pp. 22–35. With 1 map.] [**Trypanosomiasis in the Belgian Congo during 1934.**]

Eight European cases of trypanosomiasis were discovered during 1934 as compared with 12 in 1933 and 7 in 1932. In a table information is given in respect of the years 1927 to 1934 of the number of natives examined, of the number of old cases seen and treated, of the number of new cases discovered, and of the index of new infection. During the year 1934, 3,824,097 patients were examined, 86,147 old cases were seen and treated, 24,101 new cases were discovered, and the index of new infection was 0·63 per cent.

In a series of tables information is given regarding the state of affairs in the different provinces. On the whole the situation appears to be satisfactory. There is a progressive lowering of the index of new infection. van Hoof, however, records a warning that it would be unwise to draw unduly optimistic conclusions. Experience has shown that although the initial work may rapidly reduce the new infection rate, constant work is necessary to maintain the position. If vigilance is relaxed because of too great confidence in the stability of the results obtained, the disease may suddenly flare up again with the intensity of an epidemic.

A good deal of detailed information regarding the incidence and progress of the disease in the various portions of the Belgian Congo is supplied in the report, which must be consulted in the original by those interested. *W. Y.*

DUPUY. La maladie du sommeil dans les régions soumises à l'action du fonds Reine Elisabeth pour l'assistance médicale aux indigènes du Congo Belge. [**Sleeping Sickness in the Regions dealt with by the Queen Elizabeth Funds for the Medical Assistance of the Natives of the Belgian Congo.**]—*Ann. Soc. Belge de Méd. Trop.* 1936. Mar. 31. Vol. 16. No. 1. pp. 47–82.

This report describes the sleeping sickness work of the Foreami during 1934 [this *Bulletin*, 1935, Vol. 32, p. 682]. During the course of the year 744,717 natives were examined, and 4,776 new cases of trypanosomiasis discovered; the contagious index for the year was therefore 0·618 per cent.

The number of old cases on 31st December, 1933, was 12,886, and of these 2,041 were put on treatment; in all 19,703 patients were treated during the year. The total endemic index was 2·55 per cent. as compared with 1·66 per cent. in 1933. The number of provisionally cured patients was 12,975 and the efficacy rate was accordingly 65·86 per cent. A number (469) of patients disappeared during treatment, 370 died and 5,889 patients remained on treatment at the end of the year, the maintained endemic index thus being 0·76 per cent.

Having summarized the total results in this manner, Dupuy passes to an analysis of the work done in the different parts of the Bas Congo and of the two subsectors of Kwango (Bas Kwango and Bas Kwilu). In a series of tables he gives for each of the 8 sectors of the Bas Congo the contagious index, the total endemicity, the treatment efficacy rate, and the maintained endemicity rate found during each of the last four years. The author comments upon these figures.

Writing on the subject of arsenic-resistance, Dupuy says that two forms are seen. The one is acquired early during the first course of treatment; and the other is acquired slowly, or is latent, and is seen in patients who have passed their "lumbar puncture of control" [*vide* this *Bulletin*, 1935, Vol. 32, p. 683]. The latter form is by far the more dangerous because it gives rise to contagious foci which remain unknown unless one is on the look out for them. The recognition of such a danger necessitated the re-examination of patients considered to be cured in 1931. In Ganda-Sundi where 37 new cases were discovered in 1934 among 38,000 natives, systematic examination of the old cases enabled VAN DAELE to find 9 infected persons among those declared to be cured in 1931. MARSAN and ROSSI found respectively 3 cases and 12 cases belonging to the same category. The hypothesis that these old cases have been reinfected is considered and dismissed for what appear to be good reasons.

Four of the 7 foci of arsenic-treatment infections discovered in 1933 still exist. In Ganda-Sundi there were 13 cases, of which 9 were latent arsenic-resistant cases; in 1933 there were 36 cases. In Tshela-Est there were 4 cases, in one of which the resistance was acquired early; the other 3 were cases considered to be cured in 1931; in 1933 there were 35 cases. In Cataractes-Nord there were 7 primary cases of arsenic-resistance and 2 latent cases as compared with 11 cases in 1933. In Bangu there were 5 primary cases and 8 cases in which the resistance was acquired early as compared with 18 cases in 1933.

Two sub-sectors still show arsenic-resistant cases; in Seke-Banza there were two cases in which the resistance was acquired early, and in

Bas Fleuve there were 12 latent resistant infections. Arsenic-resistance is no longer found in Sengololo or in Lufimi-Basse-Sele. The total number of resistant cases found in 1934 was 53 as against 121 in 1933. It is remarked that the evolution of the disease in cases of arsenic-resistance, whether primary or acquired early in treatment, is very severe; of the 180 resistant cases recorded since 1931 in the archives of Tshele-Est, 78 cases are not cured but under control, 51 are still under treatment, and 51 are dead.

Dupuy then passes on to a discussion of methods of diagnosis and of treatment. The drugs favoured are moranyl and tryponarsyl. Arsenic-resistant cases are given a combined treatment of one or two doses of moranyl, followed by a number of massive doses of tryponarsyl, and sometimes by tartar emetic. It is noted that the massive doses of tryponarsyl (up to 4·5 gm.) are only given after a previous injection of sodium hyposulphite.

The paper closes with an account of the way in which treatment is controlled by lumbar puncture. These punctures are performed for various purposes and are designated "punctures for diagnosis," "punctures for elimination," "punctures for control," etc. The purposes for which these are made are discussed in the previous year's report [this *Bulletin*, 1935, Vol. 32, p. 683], and need not be mentioned again. It is recorded that no less than 33,317 lumbar punctures were made during 1934: the results obtained are analysed in tables.

W. Y.

i. HOARE (Cecil A.). **Morphological and Taxonomic Studies on Mammalian Trypanosomes. I. The Method of Reproduction in its Bearing upon Classification, with Special Reference to the *Lewisi* Group.**—*Parasitology.* 1936. Jan. Vol. 28. No. 1. pp. 98–109. With 1 fig. [18 refs.]

ii. ——. **Morphological and Taxonomic Studies on Mammalian Trypanosomes. II. *Trypanosoma simiae* and Acute Porcine Trypanosomiasis in Tropical Africa.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. Apr. 8. Vol. 29. No. 6. pp. 619–645. With 1 map & 2 figs. [43 refs.]

i. This paper is devoted to a critical analysis and revision of the systematics of the mammalian trypanosomes. The fact that the classification of trypanosomes has always been controversial is due mainly to lack of agreement in the choice of criteria for the identification and differentiation of the vast number of the parasites which are at present known; they are distributed among a multiplicity of hosts, and although they differ but slightly in morphology they vary considerably in their effect upon their hosts.

Practically all the known trypanosomes are referred to the genus *Trypanosoma* Gruby, 1843, with the exception of one constituting the genus *Endotrypanum* Mesnil and Brimont, 1908, and one or two others placed in the genus *Schizotrypanum* Chagas, 1909. Nothing can be said at present regarding the position of *Endotrypanum*, which is a *genus enquirendum*, its sole representative, *E. schaudinni*, requiring further investigation.

The position of *Schizotrypanum* is considered in great detail, and the author reaches the conclusion that there is no doubt that both *T. cruzi* and *S. pipistrelli* belong to the *lewisi* group of the genus *Trypanosoma* as defined in the system of HOARE & COUTELEN (1933), with additional

characteristics provided in the present paper. In his present classification HOARE divides the trypanosomes into two sections, each with distinct morphological and biological characteristics. In the section corresponding to the *lewisi* group the method of reproduction is used as a criterion for the specific differentiation of the trypanosomes.

The following is the classification :—

Family *Trypanosomidae.*
Genera *Trypanosoma.*

Definition : Flagellates which, in the course of their development in the vertebrate and invertebrate hosts, pass through the leishmanial, leptomonad, crithidial and trypanosome stages, attaining the highest degree of morphological complexity within the family.

SECTION A.

Morphology of trypanosomes in vertebrate host :

Kinetonucleus not terminal. Posterior end of the body drawn out and pointed. Division typically in the crithidial and leishmanial stages (exceptionally in the trypanosome stage).

Biological characteristics :

Multiplication discontinuous in the vertebrate host. Cyclical development in the intermediate host ; infective forms (metacyclic trypanosomes) produced in hind-gut (posterior station). Transmission contaminative (through faeces). Trypanosome slightly or not pathogenic ; easily culturable.

Lewisi group :

Reproduction by equal binary fission in the crithidial stage : *T. pipistrelli* (=*T. vespertilionis* ?).

Reproduction primarily by equal multiple fission and secondarily by binary fission in the crithidial stage : *T. rabinowitschi* (=*T. criceti*).

Reproduction primarily by unequal multiple fission and secondarily by binary fission in the crithidial stage (occasionally division in the trypanosome stage) : *T. lewisi, T. duttoni, T. primatum* ?

Reproduction primarily by binary fission in the leishmanial stage and secondarily in the crithidial stage (occasionally division in the trypanosome stage) : *T. cruzi, T. theileri* ?

Trypanosomes the method of reproduction of which is insufficiently known : *T. melophagium, T. theodori* and various other trypanosomes of rodents, insectivores, carnivores, etc.

SECTION B.

Morphology of trypanosomes in vertebrate host :

Kinetonucleus terminal or sub-terminal. Posterior end of the body blunt. Division typically in the trypanosome stage.

Biological characteristics :

Multiplication continuous in the vertebrate host. Cyclical development in the intermediate host ; infective forms (metacyclic trypanosomes) produced in the proboscis or salivary glands (anterior station). [Except in the *evansi* group in which there is no cyclical development.]

Transmission inoculative (through the bite). (Except in *T. equiperdum.*)

Trypanosomes pathogenic, not easily culturable.

Evansi group : *T. evansi, T. equinum, T. equiperdum,* etc.
Vivax group : *T. vivax, T. caprae, T. uniforme.*
Congolense group : *T. congolense, T. simiae.*
Brucei group : *T. brucei, T. rhodesiense, T. gambiense.*

ii. This paper deals with the aetiology of an acute form of trypanosomiasis among pigs in tropical Africa. Study of the parasites from a number of cases showed that they all represent a polymorphic trypanosome of the same type, *e.g.*, *T. simiae* Bruce *et al*, 1912. The following revised description of the parasite is given :—

Nomenclature :
Trypanosoma simiae Bruce *et al.*, 1912.
Synonyms : *T. ignotum* Kinghorn and Yorke, 1912.
Duttonella simiae (Chalmers, 1918).
T. rodhaini Walravens, 1924.
T. porci Schwetz, 1932.
[*T. congolense porci*] Schwetz, 1934.
T. suis Ochmann, 1905 ; Mayer, 1912.

Morphology in Vertebrate Host.—Polymorphic trypanosome, comprising (1) elongated stout forms with well-developed undulating membrane (ca. 90 per cent.), (2) elongated slender forms with slight undulating membrane (ca. 7 per cent.), and (3) short (*congolense*-like) forms (ca. 3 per cent.). The great majority of trypanosomes have no free flagellum ; it is probably present in 1·2 to 3·9 per cent. (only in elongated forms). Kinetonucleus typically subterminal and marginal. Dimensions : total length (body+flagellum) from 12μ to 24μ, mean length from 17μ to 18μ. Binary fission markedly asymmetrical (" stepped "). (*Note* : characteristic " head-to-tail " agglutination in pairs).

" *Biological characteristics.*—Intermediate hosts : tsetse flies (*Glossina morsitans* and possibly other spp.) ; development in intestine and proboscis (metacyclic trypanosomes). Pathogenicity : highly virulent to pigs and sometimes to monkeys ; slightly virulent to goats and sheep ; not infective to cattle, antelope, dogs and laboratory rodents.

" *Habitat.*—Vertebrate hosts : warthog (*Phacochoerus aethiopicus*) (reservoir) and domestic pig (*Sus scrofa*) (Suidae).

" Geographical distribution : Tropical Africa, mainly Central and Eastern."

It is shown that *T. simiae* has affinities both with members of the *congolense* group and with those of the *brucei* group, and that it occupies an intermediate position. An account is given of all the known instances of acute pig trypanosomiasis. *W. Y.*

CORSON (J. F.). **A Note on Hyrax and Dikdiks (Rhynchotragus) from Areas inhabited by Tsetse Flies.**—*Jl. Trop. Med. & Hyg.* 1936. June 15. Vol. 39. No. 12. p. 138.

Hyrax and dikdiks obtained from tsetse-free districts are known to be very susceptible to infection with *T. rhodesiense*, yet they are plentiful in places where tsetse are fairly numerous and also in sleeping sickness areas. The question arises whether these animals have acquired some resistance to infection or whether their existence in fly country depends on less exposure to infection than there seems to be.

During August, 1934, 42 Hyrax were caught in an area where *G. swynnertoni* was plentiful and trypanosomiasis among stock was troublesome, but sleeping sickness unknown. They were taken to Tinde and thick blood films were made and a white rat inoculated from each. Then in most cases the Hyrax was inoculated with the blood of a rat infected with *T. rhodesiense*. None of the blood films showed trypanosomes and none of the inoculated rats became infected. All the 29 Hyrax which lived long enough became infected. The cerebrospinal fluid of some of them showed a large number of white cells, and trypanosomes were seen in two cases.

In March and April, 1936, 42 more Hyrax were caught in the vicinity of a sleeping sickness settlement at Uyogo in the Kahama district, where *G. morsitans* was plentiful. These animals were examined and treated in exactly the same way as the previous batch, and the results were exactly the same.

The experiments on the dikdiks were similar, but on a much smaller scale, only 6 animals being used. The general conclusion is that Hyrax are probably less exposed to the bites of tsetse than casual observations of their habits would suggest. *W. Y.*

ADAMS (A. R. D.). **Trypanosomiasis of Stock in Mauritius. II.—Observations on the Incidence and Distribution of Trypanosomiasis in Cattle.**—*Ann. Trop. Med. & Parasit.* 1935. Dec. 18. Vol. 29. No. 4. pp. 475–481. With 1 map.

In 1935 the author drew attention to the fact that in addition to classical surra there existed in Mauritius a second trypanosomal disease of stock due to *T. vivax*. Since this discovery particular attention has been paid to the specific diagnosis of all trypanosomes found in domestic stock, and the present paper records the findings during the past year in respect of the incidence and distribution of these parasites in cattle.

Single blood films were then taken from the ears of local cattle selected at random from the indigenous bovine population. During the period May 1 to July 31, 1935, some 2,170 cattle were examined in this way. Of these 2·03 per cent. were found to be infected with *T. vivax* and 0·74 per cent with *T. evansi*. *T. theileri* was also recovered from several beasts. During the survey several species of filariae were observed; *Babesia mutans* and *B. bigemina* were also seen, and in a single case *Sarcocystis meischeriana* was found in a film.

Reasons are given for believing that the figure obtained for the incidence of *T. vivax* must be very much inferior to the true figure; *T. vivax* retires from the peripheral blood of cattle for weeks or months, to reappear for a few days, and again to disappear for prolonged periods.

The places in which cattle and equines infected with *T. vivax* and *T. evansi* were found are shown in a map. At first sight it would appear that while *T. evansi* has been found relatively evenly distributed throughout the island, *T. vivax* is absent from the eastern side and the south-western portion of the colony. This apparent difference in the distributions of the two parasites is explicable on two grounds. The high road with good surface roughly follows the main railway line and along it are situated the chief residential districts. The veterinary inspectors travelled on duty by rail, motor bus or cycle, and on inquiry it was found that, as was to be expected, the areas covered by them were chiefly in close proximity to the available means of rapid transport. The more inaccessible estates and villages were rarely visited. The other reason is that *T. vivax* infection in cattle did not cause the same severe and sudden disability as *T. evansi*, and consequently was not reported from all parts of the country as was the latter infection.

From a study of available data, and of the incidence and distribution of *T. vivax* at the present day, the conclusion is reached that this trypanosome may have been present in the Colony for many years. It is possible that it was present before surra was introduced in 1901.

W. Y.

SYMES (C. B.). **Outline of Work on** *G. palpalis* **in Kenya.**—*East African Med. Jl.* 1935. Dec. Vol. 12. No. 9. pp. 263–281. With 1 map. [17 refs.]

After giving a brief account of the history of sleeping sickness in Kenya, the author passes to a consideration of the present position. A map is given indicating the present distribution of tsetse fly. Many square miles of these fly-infested areas remain unoccupied by human beings. Very large areas of fertile shore and river lands lie derelict, the descendants of previous occupants of these lands maintain a miserable existence behind belts of tsetse-infested bush or mosquito-producing papyrus swamps. The lake, which should be the life and soul of all native communities within many miles of its shores, is inaccessible to the majority and a menace to the few more reckless spirits who do make use of it. The control of *G. palpalis* is, therefore, essential for the greatest of economic reasons; and whilst tsetse and infection both exist there is also the risk of more epidemics.

The next portion of the paper is concerned with the habits of *G. palpalis* in Kenya, which, Symes says, appear to differ in some ways from those of *G. palpalis* of the text books. Its breeding habits are particularly unorthodox. In Kenya very few typical breeding areas (*e.g.*, the base of rocks and trees, and under fallen logs) were found. Pupae occurred almost everywhere, in the bush, along the lake shore and river. They were discovered in relatively large numbers under a thick layer of fig leaves, with nothing but the branches of the fig tree 15 feet above the ground.

The general conclusion from this work was that it seemed impracticable to eradicate *G. palpalis* in such areas by abolishing its breeding grounds, unless by this is meant practically all the bush. Trapping experiments are being continued in three areas. Scents made from the glands of certain animals improve trapping results. It was found that *G. brevipalpis* was present in the *palpalis* bush in greater numbers than had been supposed.

The paper closes with an outline of the field trials of the "block method" of attack on *G. palpalis*, which is being financed by the Colonial Development Fund. *W. Y.*

CORSON (J. F.). **Experimental Transmission of** *Trypanosoma rhodesiense* **by** *Glossina morsitans* **from Man to Sheep and Back to Man.**—*Jl. Trop. Med. & Hyg.* 1936. June 1. Vol. 39. No. 11. pp. 125–126.

An account is given of a very interesting experiment in which *T. rhodesiense* was passed from man to a sheep by means of *G. morsitans* and then after having been transmitted through a series of six sheep by means of *G. morsitans* for a period of over a year, was passed back again to man. The scheme of transmission was as follows:—Patient (21.10.34)-fly-sheep (11.12.34)-fly-sheep-fly-sheep-fly-sheep-fly-sheep-fly-sheep (30.12.35)-fly-man (2.3.36).

A fly infected on the sixth sheep was fed on the author's arm on 2nd March; on 5th March seven rats were inoculated with the author's blood, but none became infected. Fever appeared on the 8th March, and although no trypanosomes could be found on microscopic examination of the blood, six more rats were inoculated and all became infected. Trypanosomes were found in films of the author's blood the

following day. A reaction occurred at the place of the bite on 7th March, the fifth day after the bite, and rapidly developed into a well-marked area of erythema about 2 inches across, with a darker slightly swollen and tender centre. It gradually faded and had disappeared by 17th March.

Corson considers that the local skin reaction is a useful indication of infection in persons with light skins. Its intensity depends upon the number of trypanosomes injected ; in one case when a momentary bite of a known infective fly on the finger-tip caused infection in 26 days no local reaction was seen.

This experiment supports the general opinion recently confirmed by DUKE [see below p. 657] that attacks of Rhodesian sleeping sickness treated with Bayer 205 confer no lasting immunity. This is Corson's fourth infection within 10 years, the last one being on 1st November, 1934, and 5 gm. of Bayer 205 were given between 1st November and 16th November, 1934. *W. Y.*

DUKE (H. Lyndhurst). **On the Power of** *Glossina morsitans* **and** *Glossina palpalis* **to transmit the Trypanosomes of the** *Brucei* **Group.**—*Ann. Trop. Med. & Parasit.* 1936. Apr. 8. Vol. 30. No. 1. pp. 37–38.

In a previous paper [this *Bulletin*, 1934, Vol. 31, p. 564] Duke reached the conclusion that *G. morsitans* is a more efficient vector of trypanosomes of the polymorphic group than is *G. palpalis.*

Owing to the fact that during the last year further supplies of *G. morsitans* pupae had been obtained from Tanganyika, it has been possible to extend work on these lines.

The infecting animals were monkeys, each infected with one or several strains of *T. gambiense* and *T. rhodesiense*, all of which were known to be transmissible by tsetse. Flies dying before the 10th day after the first feed were disregarded. Of 2,091 *G. morsitans* dissected 80 (3·83 per cent.) contained flagellates, whilst of 2,854 *G. palpalis* dissected 70 (2·45 per cent.) contained flagellates.

This additional evidence confirms the conclusion drawn from the earlier investigations, *viz.*, that *G. morsitans* is better able to transmit trypanosomes of the *brucei* group than is *G. palpalis.* *W. Y.*

VAN DEN BRANDEN (F.). Nouvelle contribution à l'étude de la transmission héréditaire du trypanosome. [**New Contribution to the Study of the Hereditary Transmission of Trypanosomes.**]—*Ann. Soc. Belge de Méd. Trop.* 1935. Dec. 31. Vol. 15. No. 4. p. 567.

A white rat infected with *T. brucei* gave birth to four young ones. Microscopic examination of the blood of the young rats did not reveal trypanosomes, but inoculation of their blood into a healthy rat caused infection. The author claims that he has thus demonstrated experimentally the hereditary transmission of *T. brucei* in the white rat.

W. Y.

DUKE (H. Lyndhurst) & MELLANBY (Kenneth). **A Note on the Infectivity of** *Trypanosoma rhodesiense* **in the Crop of** *Glossina palpalis.*—*Ann. Trop. Med. & Parasit.* 1936. Apr. 8. Vol. 30. No. 1. p. 39.

Many years ago, ROBERTSON [this *Bulletin*, 1913, Vol. 2, p. 242], in a paper on the development of *T. gambiense* in *G. palpalis*, drew attention

to the fact that in some cases blood may be retained in the crop of the fly unmixed and apparently unaltered for 10 days or more, although frequent feeds have intervened.

As similar observations have from time to time been made by Duke, it was decided to find out whether the trypanosomes in the crop were infective on inoculation into sensitive animals. The results of three experiments of this sort were negative, and it is concluded that the brief sojourn in the fly at about 24°C. suffices to cause the trypanosomes to lose their infectivity. *W. Y.*

ROUBAUD (E.) & COLAS-BELCOUR (J.). Essai de transmission de *Tryp. gambiense* par *Gl. palpalis* à l'Institut Pasteur de Paris. [**Attempt to transmit** *T. gambiense* **by** *G. palpalis* **at the Institute Pasteur, Paris.**]—*Bull. Soc. Path. Exot.* 1936. May 13. Vol. 29. No. 5. pp. 500–504. With 1 fig.

The flies used in this experiment were hatched from pupae sent from Entebbe. In all 100 flies were fed on the 3rd, 4th and 6th of January, 1936, on a guineapig infected with *T. gambiense* : the strain was isolated in 1934 from a case of sleeping sickness in the Cameroons. The flies were kept at about 28°C. by day and at about 35°C. by night, and the atmosphere was kept moist by dishes of water. After the infecting meals the flies were fed on a series of guineapigs up to 26th February. None of the guineapigs became infected.

The mortality among the flies was not excessive and the authors state that it was possible to keep a large number alive beyond the period necessary for the complete development of the trypanosome in the fly. All flies living 10 days or more after the infecting meals were dissected ; of the 51 so examined only one was found to be infected. The trypanosomes were limited to the gut and proventriculus. A description is given of the types of trypanosomes found ; they correspond to those previously described and figured by BRUCE, ROBERTSON and others.

W. Y.

LLOYD (H. M.). **Notes on the Bionomics of** *Glossina swynnertoni*, **Austen.**—*Bull. Entom. Res.* 1935. Dec. Vol. 26. Pt. 4. pp. 439–468. With 5 figs. [10 refs.]

The observations and experiments upon which this paper is based were made in Shinyanga, Tanganyika Territory, from December 1930 to July 1932. Their subject, *G. swynnertoni*, an important vector of both human and animal trypanosomiasis, was, prior to the commencement of reclamation, rapidly extending its area in the Shinyanga district and forcing the native inhabitants to retire.

A description is given of the four main types of vegetation communities infested by this fly round Shinyanga ; in each type game in moderate amount, and therefore food, is present throughout the year. While most common in " hard pan " (valleys where the surface soil lies just above beds of " an impervious limey concrete " or pseudo-laterite rock), *G. swynnertoni* is much less abundant in country in which the dominant tree is *Commiphora fischeri* ; scarce in open acacia savannah ; and of still less frequent occurrence in open plains. This species of tsetse breeds mainly in thickets, but its " pupae are also found under decumbent logs, overhanging rocks and in hollows at the bases of trees." As regards seasonal prevalence, " *G. swynnertoni* is commonest during

the first month or two of the dry season," becomes less numerous with the advance of the latter, and sinks to its lowest level during the short rains. An increase occurs in the short dry season (in January or February) following the short rains, and thenceforward, throughout the long rains, numbers show little alteration. Owing to the presence of game, " concentrations remain in the hard pan areas throughout the dry season."

Experiments showed that a clearing 800 yards wide is not an absolute barrier to *G. swynnertoni*, which, like *G. morsitans*, " appears to find food by sight rather than by scent." Flies that cross such a clearing are mostly hungry, and it is believed they are searching for food. The hungrier the fly the more persistently does it follow man, and in this respect males exhibit more pertinacity than females. Catching *G. swynnertoni* by hand from screens, in a block of country some 15 square miles in extent during a period of sixteen months, reduced its numbers but did not exterminate the fly. The failure of the experiment in the latter respect is ascribed by the author to the insufficient width of the isolating clearings, and also to the fact that female flies show themselves to catchers less frequently than males, " so that breeding is able to continue." In the first months of the experiment the catch was predominantly male ; subsequently " A very large rise in female percentage occurred." *E. E. Austen.*

i. LAMBORN (W. A.) & HOWAT (C. H.). **A Possible Reservoir Host of** *Trypanosoma rhodesiense.—Brit. Med. Jl.* 1936. June 6. pp. 1153–1155.

ii. DAVEY (J. B.). **Trypanosomiasis.** [Correspondence.]—*Ibid.* June 27. pp. 1321–1322.

i. Details are given of a Nyasaland native infected with *T. rhodesiense*, but showing no signs of the disease.

The patient was admitted to hospital at Fort Johnston on 16th June, 1935, suffering from urinary schistosomiasis and ankylostomiasis. On examination of his blood polymorphic trypanosomes (*T. rhodesiense*) were found. The native has been kept under observation for over 6 months, and apart from an improvement in his general health as a result of treatment of the hookworm and bilharzia, the condition remains unchanged. Except for rises of temperature on two occasions the course has been afebrile ; he is content to remain in hospital and weaves mats, the intricate nature of this work indicating that his muscular co-ordination is unimpaired.

The blood has been examined at weekly intervals and the number of trypanosomes found per 100 leucocytes is given in a chart. Once every month the condition of the cerebrospinal fluid and of the lymph has been investigated, but without pathological findings. The trypanosome was transmitted to dogs and rats by the syringe and by the fly ; it produced in these animals an acute disease.

The authors state that it is apparent that this patient is either passing through a latent stage in the course of active trypanosomiasis or else that he is a carrier of the disease. Since the patient has so long remained able-bodied, and since he resides in an area infested with *G. morsitans*, it is evident that any flies which might have bitten him could well have passed on the infection to others, and that in this way could have been started an epidemic comparable to those which have been traceable to one infected person. The history of the first outbreak

of sleeping sickness in Nyasaland is particularly instructive in this respect, and is referred to in some detail.

From the sleeping sickness diaries, it appears that Dr. J. B. Davey in 1908 diagnosed the first case of sleeping sickness found in the Protectorate in a native at Nkata Bay, who had accompanied a European to Tanganyika and the Congo. The native returned to Nyasaland from Cape Town by steamer. Davey records that the patient was in good health, the glands were not greatly enlarged and examinations of the blood were negative, and that the native had been back in his village, in a fly-free area, for one year, during which time he had made no complaint of ill health.

The patient was removed and segregated near Dowa about 130 miles south. His removal in a condition comparable to that of the native, who is the subject of the present communication, and the want of proper care to shield him from fly, appears to have been responsible for the epidemic of sleeping sickness which followed in the Dowa district.

The conclusions are as follows :—

"This case may well afford an affirmative answer to the question : Can *T brucei*, occurring in areas where human trypanosomiasis is unknown, infect man ? Its importance lies in the fact that the spread of sleeping sickness is, in all probability, mainly due to the movements of human carriers of a strain of trypanosome which, while not immediately pathogenic, may become so after more than one passage through man.

"It emphasises the need for examination of the whole population when sleeping sickness arises apparently *de novo*. The investigation should include the examination of the blood of even the apparently healthy and of material obtained by puncture of the lymph glands."

ii. Davey, referring to Lamborn and Howat's paper, states that if, as these authors believe, the trypanosome on first transfer to man from game, *via* the fly, establishes itself in its new host with difficulty, and even then is only mildly pathogenic, we have an explanation of the escape from infection of large numbers of persons bitten by infected flies. There still, however, seems to be a hiatus between the discovery of a carrier by Lamborn and Howat and the usual acute course of infection. Surely one passage through man does not so intensify the pathogenicity of *T. brucei* to man that it becomes capable of causing death in 100 per cent. of untreated cases within 4 months. Davey suggests that this hiatus does not, in fact, exist. He refers to two cases reported by the Medical Officer in charge of sleeping sickness investigations, 1917, which long survived the usual 4 months' duration of the disease. There are indeed good grounds for believing that one of them actually recovered, and the other showed no signs of disease 8½ months after a diagnosis had been made. Davey agrees with Lamborn and Howat that the human carrier is the danger. If infection of man may arise in any area where *G. morsitans* and game harbouring *T. brucei* exist, as recent knowledge of the disease suggests, Davey enquires whether the time is not ripe for reconsidering the conclusions to which the Royal Society's Commission to Nyasaland came. One of these reads :

"It is self-evident that these wild animals should not be allowed to live in 'fly-country,' where they constitute a standing danger to the native inhabitants and the domestic animals. It would be as reasonable to allow mad dogs to live and be protected by law in our English towns and villages. Not only should all game laws restricting their destruction in 'fly-country' be removed, but active measures should be taken for their early and complete blotting out."

W. Y.

SICÉ (A.), ROBIN (Ch.) & MERCIER (H.). Forme évolutive atténuée de la trypanosomiase humaine ; ses rapports avec un trypanosome de moindre virulence. [**Attenuated Form of Human Trypanosomiasis ; its Relationship with a Trypanosome of Slight Virulence.**] —*Marseille-Méd.* 1936. Mar. 15. Vol. 73. No. 8. pp. 345–351. With 1 fig.

The authors have studied the morphology of a trypanosome isolated from a very chronic case of human trypanosomiasis.

The history of the patient is given in considerable detail. He left the Ivory Coast for France in July, 1934, apparently in a state of perfect health, and remained well until April, 1935, when he was admitted to hospital with pain in the legs, a bilateral oedema, general adenitis and tachycardia. Trypanosomes were found on gland puncture. Lumbar puncture showed slight alterations in the spinal fluid, the cells being 24 per cmm. and the protein 0·30 per 1,000.

After some difficulty a guineapig was infected with the trypanosome, and then the strain was transmitted through a series of guineapigs. Attempts to infect mice failed. The authors remark upon the long incubation period, the prolonged infection and the absence of abortion in the infected guineapigs. They state that by observing daily the evolution of the trypanosomes in the infected animals they have been able to follow the adaptation of the parasite for the organism of the guineapigs.

The trypanosome at the beginning was remarkably polymorphic and it still retains the character, but to a less degree. The multiplication of the trypanosome did not appear to the author to be particularly active, but it exhibited interesting characters. Some forms were noted in which the nucleus had divided, and one nucleus was near the undivided blepharoplast and the other at the junction of the middle and posterior thirds of the body.

The authors write that the trypanosome in the course of its adaptation to the guineapig presented most of the various characters exhibited by *T. rhodesiense*, *e.g.*, marked polymorphism and posterior-nuclear forms. These points are illustrated in a text-figure. The patient, however, suffered from a chronic disease and came from a region, the Ivory Coast, where Rhodesian sleeping sickness is unknown. *W. Y.*

MOUSTARDIER (G.). A propos d'un cas de méningo-encéphalite trypanosomique chez un Sénégalais à Tananarive. [**A Case of Trypanosomal Meningo-Encephalitis in a Senegalese at Antananarivo.**]—*Bull. Soc. Path. Exot.* 1936. Apr. 1. Vol. 29. No. 4. pp. 435–442.

The patient was a native of the Ivory Coast who enlisted for 4 years in February, 1929. He served for 3 years in Senegal and was repatriated eventually to the Ivory Coast. In February, 1933, he re-enlisted for a further period of 4 years and left Africa on August 31, 1933, for France whence he was sent to Madagascar where he arrived on October 26, 1933. The patient was admitted to hospital at Antananarivo owing to an epileptiform attack in August, 1935. The findings and subsequent history of the case are given in great detail.

The case turned out to be one of trypanosomal meningo-encephalitis in an advanced stage. Spinal puncture revealed trypanosomes, lymphocytosis and an excess of globulin. The date of infection is

unknown, but it could not be later than August, 1933, when he left West Africa for France. The disease advanced insidiously for two years without producing general symptoms; there were no febrile disturbances, no emaciation and no somnolence. *W. Y.*

VAN BOGAERT (Ludo). Présence de lésions myéliniques dans la trypanosomiase expérimentale. [**Presence of Myelin Lesions in Experimental Trypanosomiasis.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 13. p. 1387.

In a previous paper the author with BORREMANS drew attention to the occurrence in a case of human trypanosomiasis of a cystic degeneration of the subthalamic region and in the neighbourhood of the third ventricle. There was a micro-polycystic degeneration of the parenchyma involving the myelin fibres.

More recently the author has had the opportunity of examining the brain of a baboon (*Papio jubilaeus*) infected experimentally with *T. gambiense*. He found definite involvement of the myelin of the centrum ovale and of the claustrum by foci irregularly distributed round the vessels. In addition to these juxta-vascular foci, there was a marked demyelinization of the corona radiata, and particularly of the fibres which go to the corpus callosum. The myelin lesions were old, and the demyelinized areas were the seat of very manifest macro- and micro-glial proliferation. The myelin lesions were accompanied by very gross lesions of the axone. *W. Y.*

BOUVIER (G.). Le diagnostic microscopique des trypanosomiases bovines en brousse. [**Microscopic Diagnosis of Trypanosomiasis in Cattle in the Bush.**]—*Bull. Agric. Congo Belge.* 1936. Mar. Vol. 27. No. 1. pp. 65–69.

This paper describes the various methods of examining the blood of cattle for trypanosomes and contains nothing new. *W. Y.*

DUKE (H. Lyndhurst). **On the Prophylactic Action of "Bayer 205" against the Trypanosomes of Man. Concluding Observations.**—*Lancet.* 1936. Feb. 29. pp. 463–469. [12 refs.]

In this paper the author concludes his observations on the prophylactic action of Bayer 205 against human trypanosomiasis [this *Bulletin*, 1935, Vol. 32, p. 8].

Referring to his former paper, Duke writes that the use for the first time of native volunteers on a large scale involved difficulties that have since been largely overcome, and explained the inclusion in his first paper of the men of Group I, *i.e.*, infected cases treated with Bayer 205. There were, of course, objections to their employment, *i.e.*, the large amount of Bayer administered and the possible immunizing effect of the liberation of antigen following the destruction of trypanosomes by the first dose. The full extent of the protection conferred by the drug against *T. rhodesiense* was not determined, as no protective volunteers succumbed to that trypanosome.

In the course of the present investigations several of the volunteers after having been infected and treated were later on again exposed to infection. Details of this work are given in a table, from which it appears that any immunity conferred by a brief (10 to 15 days) infection

with *T. rhodesiense* or *T. gambiense*, followed by a course of six injections with Bayer 205 or tryparsamide, does not persist for as long as 6 to 16 months.

In the second table information is given regarding the observations made on 18 further volunteers. Nine received a single dose of 1 gm. of Bayer 205, five a single dose of 2 gm., and the other four 2 doses of 1 gm., separated by an interval of 21 days. All the injections were given intravenously. Of the nine who received 1 gm. of the drug four were infected at their first exposure, *viz.*, 92, 105, 92 and 73 days, respectively, after the administration of the drug. Two of the infections were *T. gambiense* and two *T. rhodesiense*. The remaining five were protected for 120, 123, 97, 190 and 327 days, respectively. Of the volunteers receiving 1 gm. + 1 gm. of Bayer 205, none were infected at their first exposure. Protection against *T. gambiense* lasted 169 and 187 days, and against *T. rhodesiense* 171, 206 and 164 days. None of those receiving 2 gm. of the drug were infected at their first exposure, and protection against *T. gambiense* lasted 128 and 153 days, and against *T. rhodesiense* 163, 103, 152, 180 and 179 days.

As a general rule when infection did ensue it developed normally. There were, however, certainly three, and possibly five, examples of delayed or " cryptic " infection, when the patient showed no apparent symptoms for two months and possibly longer. An infection of this kind may gradually generate typical symptoms, or it may become merged into a subsequent infection superimposed upon it and running a normal course.

The matter is discussed in considerable detail, and Duke reaches the conclusion that " cryptic " infection may arise independently in nature, apart altogether from the administration of any drug.

Consideration of one of the cases suggests that frequently repeated inoculations of living trypanosomes during the three or four months immediately following the administration of Bayer 205 lead to the establishment of a more prolonged immunity than that conferred by the drug alone without such frequent exposures to infection. Duke writes that if this be true, then the more intense the exposure in nature to infective tsetse, the greater the benefit derived from the prophylactic.

The general conclusion is that a dose of 2 gm. of Bayer 205 administered to an adult may be expected to confer protection against *T. gambiense* and *T. rhodesiense* for at least three months ; and the protection may last longer.

W. Y.

VICARS-HARRIS (N. H.). **The Occupation of Land reclaimed from the Tsetse Fly of Tanganyika.**—*East African Ann. 1934–35.* Nairobi. 6 pp. With 10 figs. [Summarized in *Rev. Applied Entom.* Ser. B. 1936. May. Vol. 24. Pt. 5. pp. 103–104.]

" A brief account is given of the land that has been reclaimed from *Glossina* in Tanganyika Territory, particularly in the Shinyanga district, and of the means adopted to induce permanent settlement by natives. Between 1923 and 1931 some 140 square miles of country had been reclaimed by the natives in Shinyanga under the guidance of the Government ; between 1927 and 1932 some 80 square miles were reclaimed in Mwanza Province, and a smaller area in Nzega was cleared and closely settled. In the Mbulu District in the Northern Province, over 200 square miles was made available in 1931 by cutting corridors and throwing open hitherto inaccessible plains. A rather more

detailed account is given of the opening up of the Huruhuru plains for grazing and settlement. Unfortunately the corridor 1,200 yards wide leading to these plains was found to be too narrow for safety, as small numbers of flies were observed wandering into it and along the cattle track. Ultimately it is hoped to render free from fly an area three or four miles wide, but in the meantime cattle were successfully taken through by sending trained native assistants with catching screens ahead of each herd. Not many flies were caught. An area of 150 square miles has been opened up, and the sojourn of cattle in it during the first season has been a great success."

YORKE (Warrington) & MURGATROYD (F.) in collaboration with F. GLYN-HUGHES, H. M. O. LESTER & A. O. F. ROSS. **A New Arsenical for the Treatment of Syphilis and Trypanosomiasis.**—*Brit. Med. Jl.* 1936. May 23. pp. 1042–1048.

During the last seven or eight years, the authors have examined for the Chemotherapy Committee of the Medical Research Council the trypanocidal activity of numerous new compounds, including a long series of aromatic arsenicals of the general formula—

prepared in Professor MORGAN's Laboratory of the Department of Scientific and Industrial Research at Teddington. The initial steps in this investigation consisted in examining the trypanocidal activity of the compounds first in mice and later in rabbits infected with a standard trypanosomal infection, the response of which to old and well-tried drugs, such as atoxyl and tryparsamide, had been thoroughly investigated.

About half a dozen of these new compounds were found to have a therapeutic activity in trypanosome-infected mice at least equal to that of tryparsamide. One of the most active of these, "Neocryl," was selected for more extensive trial on larger animals, and eventually on man. This product, which is to be put on the market as "Crylarsan Brand of Neocryl," is sodium succinanilomethylamide-p-arsonate. It is a white crystalline substance readily soluble in water.

The drug was tried on a considerable number of patients suffering from various stages of syphilis and on a limited number of cases of Nigerian sleeping sickness. The portion of the paper relating to the trials on syphilis has been summarized in the *Bulletin of Hygiene* (1936, Vol. 11, p. 693) and does not require further notice here. The trials on Nigerian sleeping sickness were carried out by Dr. Lester, Deputy Director of the Tsetse Investigation in Nigeria. The results obtained are summarized in the following table :—

It is stated that " the stimulant action of a long course of treatment with the drug was very pronounced, and led to the same difficulty commonly met with in experimental work with tryparsamide. Patients felt so much better that they became tired of the prolonged treatment, and absconded before they had completed their course of injections. Of the first eight cases treated with neocryl five disappeared before the final examination of the cerebro-spinal fluid could be made. In every case patients showed very rapid clinical improvement, and thought themselves completely cured after they had had five or six injections.

TABLE IV.—*Summary of Results Obtained in the Treatment of Cases of Nigerian Sleeping Sickness with a Single Course of Neocryl.*

Case No.	Duration of Symptoms	Condition at Commencement of Treatment					Treatment		Condition Immediately after Treatment			Remarks
		Clinical	Blood Tryps.	Glands Tryps.	Cerebro-spinal Fluid		Dates	Total Amount of Neocryl Given	Clinical	Cerebro-spinal Fluid		
					Globulin	Cells				Globulin	Cells	
4503	3 months	Weakness, severe headache, sleeplessness at night, sleeping during day, adenitis	+	+	+	28	28/6/34 to 18/9/34	28 grams (2 gram doses)	Quite strong, no headache, sleeps well at night, not in daytime; adenitis gone, blood negative	+	219	
4521	2 ,,	Weakness, headache, loss of weight, somnolence, adenitis	+	—	++	238	7/7/34 to 24/9/34	27 grams (2 gram doses)	Quite strong, no headache, sleeps normally, put on weight; no adenitis, blood negative	Slight haze	39	
4510	1 year	Weakness, very severe headache, somnolence, dullness, slurring speech, emaciated	+	—	+++	332	5/7/34 to 24/9/34	25 grams (2 gram doses)	Quite strong, no headache, sleeps normally, appears and looks perfectly normal; no adenitis, blood negative	—	1	
B.K.	6 months	Weakness, severe headache, somnolence, dullness, some adenitis, well nourished	+	—	Normal	Normal	26/6/34 to 9/8/34	15 grams (2 gram doses)	Quite strong, no headache, sleeps normally; no adenitis, blood negative			Absconded 12/8/34
K.S.	1 month	Weakness, somnolence, slight adenitis	+	—	,,	,,	26/6/34 to 15/9/34	29 grams (2 gram doses)	Quite strong, sleeps normally; no adenitis, blood negative			Absconded 15/9/34
A.G.	6 months	Weakness, headache, somnolence, dullness, loss of weight, some adenitis	+	+	+	42	30/6/34 to 23/8/34	19 grams (2 gram doses)	Quite strong, no headache, sleeps normally, blood and gland juice negative			Absconded 24/8/34
M.D.	6 ,,	Headache, well nourished, adenitis	—	+	Normal	Normal	10/7/34 to 9/8/34	11 grams (2 gram doses)	No headache, feels quite well, blood and gland juice negative			Absconded 12/8/34
K.G.	4 ,,	Weakness, severe headache, somnolence, some adenitis	—	+	+	128	10/7/34 to 26/9/34	29 grams (2 gram doses)	Stronger, no headache, no somnolence, able to do light work, blood and gland juice negative			Absconded
S.J.	6 ,,	Weakness, headache, somnolence, dullness, adenitis; history of syphilis six years previously and of "eye trouble" two months previously	+	+	+	70	9/3/35 to 30/4/35	31·5 grams (3 gram doses)	Quite strong, no somnolence, appears and feels perfectly normal; blood and gland juice negative	—	3	
1	3 ,,	Weakness, headache, fever, somnolence, dull, emaciated	—	+	++	296	?	28·5 grams (3 gram doses)	Improved, but still somnolent	+	150	
2	4 ,,	Weakness, headache, fever, emaciated	+	+	+	21	?	28·5 grams (3 gram doses)	Normal	—	0	

"Taken as a whole, the results were distinctly promising. No late cases were included in the series, but it is noteworthy that one very advanced case that had failed to react to full courses of tryparsamide, of Bayer 205, and of antrypol, for some reason or another improved markedly on being given a course of this new drug." *W. Y.*

KELLERSBERGER (E. R.). **African Sleeping Sickness : a Clinical Study.** —*Southern Med. Jl.* 1936. Mar. Vol. 29. No. 3. pp. 239–242. With 3 figs.

This paper gives an interesting summary of the author's work on the treatment of sleeping sickness at Bibanga in the Katanga Province of the Belgian Congo.

Since 1924, when the present small hospital was completed, over 40,000 patients have been seen, and about 11,000 (28 per cent.) of these had sleeping sickness. It is noted that most of the patients presented themselves voluntarily, and that about 75 per cent. were observed for two years or more, and some for as long as 5 or 10 years. The greatest number of cases discovered was 2,915 in the year 1927.

The patients were divided roughly into three groups. The first group comprises those in apparent good health with trypanosomes in the peripheral blood, with irregular fever and persistent headaches, and inability to do full work. The second group comprises cases which present definite clinical signs, such as adenitis, constant headaches and irregular fever, tremors, slow slurring speech, stupor, and slow mental response. Gland puncture in these cases usually revealed the presence of the parasite ; and lumbar puncture often showed increased pressure, excessive globulin and lymphocytosis. No less than 75 per cent. of the patients fell into this group. The last group comprises all advanced cases, with varied symptoms, such as somnolence amounting almost to coma, incoordination of movements, occasional involuntary defecation and urination, and sometimes mild or violent insanity. This group contained 13 per cent. of the patients.

Atoxyl, antimony, germanin and tryparsamide were the four drugs used. Kellersberger states that a number of patients were cured with atoxyl, some of whom had been observed for over 7 years. The disadvantages of atoxyl are the danger of blindness, the need of prolonged treatment, consisting of repeated series of injections over several years, its uselessness in advanced cases, and the difficulty of persuading a native to submit to painful injections over a long period.

Antimony preparations were never used alone, but always in conjunction with tryparsamide, when there was concomitant schistosomiasis, or when tryparsamide alone did not appear to be efficient.

Bayer 205 was extensively used, over 4,000 injections being given intravenously in an average dose of 0·02 cgm. [gm. ?] per kilo. of body weight. Short courses of 3 or 4 injections cured early cases where the trypanosomes were essentially a blood parasite. All the cases of the second or third group, however, relapsed after temporary improvement, and then had to be treated with tryparsamide.

In the author's opinion, the most useful drug for all cases is tryparsamide. Since 1924, it has been the drug of choice in the Congo, and no less than 150,000 injections have been given by the native staff to 10,000 cases. To effect a cure the drug must be pressed to the limit of security. The average dose was 0·045 gm. per kilo. ; anything in excess of this may cause blindness, especially in advanced cases. In

itinerant work in the bush, where it is difficult to control the patient, it is wiser to use small doses for longer periods, and consequently the itinerant personnel of the Belgian Government never exceeds a 2 gm. dose. Patients who weigh 70 kilos. or more received in the author's dispensary as much as 3 gm. or even 4 gm. per week with excellent results. It was found that a series of 15 injections weekly is preferable to the 8 or 9 given formerly, and in cases where the central nervous system is involved it is wiser to repeat the cure after a rest of from one to three months, depending upon the condition of the patient as judged by lumbar puncture and other examinations.

Tryparsamide, as a rule, cures cases of Group I, but it is in the more advanced cases that it is the most efficient of all drugs. Patients who were moribund and apparently hopelessly insane have been cured, and the results have been controlled in many cases for 6 or 7 years. Kellersberger states that he has considered as cured a patient who is in good clinical condition one year after treatment and with negative laboratory examinations. As many as 75 per cent. have been cured, and under ideal conditions it is possible to obtain an 80 or 90 per cent. cure.

W. Y.

VAN DEN BRANDEN (F.), APPELMANS (M.) & POTTIER (R.). La toxicité du tryponarsyl additionné d'hyposulfite sodique pour le parenchyme rénal. [**The Toxicity of Tryponarsyl with Sodium Hyposulphite for the Renal Parenchyma.**]—*Ann. Soc. Belge de Méd. Trop.* 1936. Mar. 31. Vol. 16. No. 1. pp. 121–123.

For some time it has been considered advantageous to associate sodium hyposulphite with tryponarsyl in the treatment of sleeping sickness in the Congo ; the sodium hyposulphite is believed to lessen the risk of visual disturbance resulting from the arsenical.

The authors have investigated the question whether sodium hyposulphite increases the general tolerance of the rabbit to tryponarsyl, and have reached the conclusion that it does not do so. In the second part of their work they examined particularly the kidneys of rabbits which had received various doses of tryparsamide alone, and of other rabbits which had received the same doses of this arsenical and, in addition, various doses of sodium hyposulphite. The kidneys of the rabbits which had been given tryparsamide alone (up to a dose of 1·25 gm. per kilo.) showed no pathological changes, whereas the kidneys of the rabbit which had been given 1·25 gm. of tryparsamide+0·375 gm. of sodium hyposulphite per kilo. showed severe lesions. The conclusion is that sodium hyposulphite does not protect the renal epithelium from damage by tryparsamide, but, on the contrary, may increase the risk.

W. Y.

POTTIER (R.) & VAN DEN BRANDEN (F.). Contribution à l'étude toxicologique de la glyphenarsine (tryparsamide, tryponarsyl, novatoxyl, trypotan). [**Contribution to the Study on the Toxicity of Glyphenarsine (Tryparsamide, Tryponarsyl, Novatoxyl, Trypotan.**] —*Ann. Soc. Belge de Méd. Trop.* 1936. Mar. 31. Vol. 16. No. 1. pp. 105–107.

The substance of this paper has already been published elsewhere and noticed in this *Bulletin* [1936, Vol. 33, p. 201]. *W. Y.*

STEWART (J. L.). **Treatment of Trypanosomiasis by Tartar Emetic, Antimosan and "Surfen C" in the Gold Coast.**—*Jl. Comp. Path. & Therap.* 1935. Dec. 31. Vol. 48. Pt. 4. pp. 316–318.

A summary is given of the result of treating with antimony, and a new Bayer preparation "Surfen C," Gold Coast cattle suffering from trypanosomiasis.

The cattle of the Gold Coast differ from those of Nigeria. The latter are humped zebus, while the former belong to the West African Shorthorn unhumped breed, which has a much higher resistance to trypanosomiasis than the zebu.

Details of the result of treating cattle belonging to both types are given. The treatment by "Surfen C" did not appear to be nearly as effective as that by tartar emetic and other antimony preparations, but "Surfen C" appeared to be useful after antimony treatment to prevent relapses. All the 85 cattle are alive, except two, which died from intercurrent infections. The zebu bullocks which relapsed are, however, in very bad condition, but the zebu cows and heifers treated by antimony appear to have made a complete recovery and are in excellent condition one or two years after treatment. *W. Y.*

OESTERLIN (M.). Zur Chemotherapie der Infektionskrankheiten. [**On the Chemotherapy of Infectious Diseases.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1936. May 14. Vol. 118. No. 3. pp. 263–306. With 1 fig. [66 refs.]

This long and rather technical paper must be consulted in the original by those interested. It is shown that chemotherapeutic action is dependent upon the optical activity of a substance, and that both antipodes behave very differently against trypanosomes, just as the capacity to dye animal or vegetable threads by the same substances differs.

A series of mice were given 20 mgm. of casein-atoxyl compound on 1st October, and nine days later were infected with *T. brucei*. On the 13th October the mice were treated with various doses of atoxyl, and it was found that 0·8 mgm. sufficed to produce a permanent cure. In mice which had not received the preliminary dose of casein-atoxyl compound the maximum dose of 4·0 mgm. of atoxyl failed to produce a complete cure. This means that the effective dose of atoxyl is reduced to 20 per cent. when, as the result of a preliminary treatment of the experimental animal, the corresponding antibody has been produced. The phenomenon is strictly specific; previous administration of casein-atoxyl does not reduce the curative dose of tryparsamide or spirocide.

The toxicological and direct-acting factor in the metal-free derivatives of acridin and chinolin is bound up with the fluorescence of these substances. The fluorescent emissions of these substances were spectrographically absorbed and the relationship between fluorescence and activity ascertained. The activity of substances is only developed when there is a firm anchoring to the parasite cell. As a result of this firm binding the optical properties are changed; the exciting radiation is turned in the visible direction and the fluorescent colour is altered. In a table the author gives details regarding the colour in neutral solution, the fluoresence in neutral solution, the fluoresence in solution pH 6, the fluoresence in the trypanosome in ultra violet and in white light, and the trypanocidal activity in respect of 23 acridin and chinolin

derivatives. It is noted that the fluorescence seen in the trypanosomes can be approached *in vitro* by adjusting the pH.

Fluorescent substances, such as rivanol, which possess no binding groups are inactive. It is suggested that in the trypanocidal acridin and chinolin the pentavalent nitrogen serves as the haptophore groups ; rivanol, when changed into rivanol-methylsulphate, becomes trypanocidal.

Chemotherapeutic interference is not a biological but a pure physico-chemical phenomenon due to the occupation of the trypanosome receptors by feebly or completely inactive products, thus preventing the specific binding of active substances.

Experiments on interference have produced evidence that all such substances as the arsinic acids, chinolin and acridin compounds are anchored by the same constituents of the trypanosome cell. *W. Y.*

SAZERAC (R.) & LARTHE (N.). Action de certains dérivés du cuivre sur le *Treponema cuniculi* et le *Trypanosoma brucei*. [**The Action of Certain Derivatives of Copper on** *Treponema cuniculi* **and** *Trypanosoma brucei*.]—*C. R. Soc. Biol.* 1935. Vol. 120. No. 40. pp. 1179–1181.

Copper oxide and hyposulphite were tried on rabbits infected with *Trep. cuniculi*. The former, which was given subcutaneously in oily suspensions, produced immediate benefit, but relapse followed ; the latter given intravenously gave better results, and definite cures were obtained with two injections of about 14 mgm. per kilo.

Perla (1934) has shown that copper sulphate given by the mouth for 12 days protects to some extent rats against subsequent infection with *T. lewisi* or *T. equiperdum*. The authors examined the action of oily suspensions of copper protoxide in rats infected with *T. brucei*. It was found that a dose of 20 mgm. per 100 gm. sufficed to clear the circulation temporarily. A pseudo-colloidal solution of copper (electrocuprol) likewise exerted a definite temporary action, as also did copper sulphate.

W. Y.

SCHILLING (Claus) with H. SCHRECK, H. NEUMANN & H. KUNERT. Versuche zur Schutzimpfung gegen Tsetsekrankheit. III und IV Teile. [**Experiments on Protective Inoculation against Tsetse Disease. Parts III and IV.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1936. Jan. 9 & Apr. 15. Vol. 87. Nos. 1/2 & 5/6. pp. 47–71 ; 482–518. With 19 figs. [12 refs.]

In these two lengthy papers the authors continue the account of their work on immunization against trypanosomes [this *Bulletin*, 1935, Vol. 32, p. 43 and p. 714, and 1936, Vol. 33, p. 195].

The third communication deals with the antigen properties of various trypanosome species and strains. In this work the authors employed, in addition to certain old strains collected between 1911 and 1914, various strains of *T. congolense*, *T. brucei* and *T. rhodesiense* of comparatively recent date. A list of these strains is given and information is also supplied regarding the manner in which they have been maintained. The antigenic property and the receptor (sensitivity to antibody) are two different attributes of the trypanosome cell. Experiments to illustrate the truth of this are given. From a strain of " Ferox " a serum-fast variety was obtained. Mice were infected with

the normal strain and then cured ; some were then inoculated with the homologous normal strain, and others with the serum-fast strain ; the former were refractory, but the latter became infected. When, however, mice were infected with the serum-fast strain and then cured they were found to be resistant to both the normal and the fast strain. This experiment shows that the serum-fast strain had the same antigenic qualities as the normal strain, but not the same receptors.

It was found that genuine strains of a trypanosome species obtained from several places in East Africa might behave similarly or differently in respect of their antigenic property and receptors. Such changes—loss of antigenic property or of receptors—may be produced in one of the following ways :—(*a*) during an infection—relapse strain formation ; (*b*) as the result of previous treatment with dry trypanosomes ; (*c*) as the result of the passage of the strain through Glossina ; and (*d*) by the transference of a strain from one species of animal to another.

When a second infection is superimposed on a first, the antibodies to the first infection may disappear from the serum and antibodies to the second infection may appear. Antibodies appear in different animals at very different times after infection ; in the foal Lottchen they appeared on the 126th day, in the foal Zeus on the 49th day, and in a rabbit on the 9th day.

In order to test the value of sera in respect of parasiticidal antibodies, the author always used the *in vivo* mice experiment as a standard. The serum, immediately after it was obtained, was always heated to 45°C. for 30 minutes to kill any trypanosomes it might contain. Sufficient trypanosomes were then added so that the suspension when examined microscopically showed not more than one trypanosome to a field ; 0·5 cc. of this suspension was then injected intraperitoneally into mice. If the suspensions contained substantially more trypanosomes the trypanocidal action was not manifest, or of 2 mice injected only one failed to become infected. Sometimes parallel *in vitro* observations were made, but these did not always agree with the *in vivo* results. The authors, however, did not succeed in establishing that there existed any real difference between the *in vitro* antibodies and the *in vivo* antibodies.

Discussing the question whether in trypanosomiasis a spontaneous sterilizing immunity ever occurs, the authors give details of two cases in which it seemed probable, but it is not possible to speak with certainty on this matter. [This should be compared with VAN SACEGHEM's findings, see below.]

It was found that the parasiticidal antibodies of the serum could pass from the mother to the offspring.

It is also noted that the strain of *T. brucei* " Hamburg alt," which in 1914 developed in Glossina without difficulty was no longer capable of doing so in 1933.

RONDONI and Schilling (1913) showed that a trypanosome suspension kept at 37°C. for some hours (which killed all the parasites) was toxic when injected into mice. In 1930 Schilling and Neumann repeated this work with the same strain of trypanosomes and failed to confirm the previous observation. It was found, however, that suspensions of a strain of *T. brucei* (maintained two years in mice), and kept at 37°C. for 18 hours was exceedingly toxic for mice.

The fourth communication deals more generally with trypanosome infections. It is difficult to follow closely the course of an infection in the larger experimental animals. When the number of trypanosomes

in the blood is small subinoculation into laboratory animals does not always result in infection. This is the basis of the method of immunization by minimal infection. A single cell infection with genuine trypanosome strains does not succeed; and the number of parasites injected by the bite of Glossina is not a matter of indifference. The species of trypanosomes (*T. brucei*, *T. congolense* and *T. vivax*) exhibit considerable differences in infectivity. An account is given of the characters of these parasites as seen in the vertebrate and invertebrate hosts, but it contains nothing new.

It was found that trypanosome infections in pregnant cows usually cause abortion; the infection did not pass to the foetus.

If an infected Glossina sticks its proboscis into the epidermis only, no infection results; infection does not occur unless the proboscis penetrates to the cutis and enters a blood vessel.

When cattle have a double infection with *T. brucei* and *T. congolense* one infection usually causes suppression of the other; in general *T. congolense* is the stronger. In such mixed infections *T. brucei* is the more easily eliminated by treatment. In double infections with *T. lewisi* and *T. congolense* the parasites exert no influence upon one another. *W. Y.*

VAN SACEGHEM (René). L'immunisation des bovidés contre la trypanosomiase. [**The Immunization of Cattle infected with Trypanosomiasis.**]—*Bull. Agric. Congo Belge.* 1936. Mar. Vol. 27. No. 1. pp. 47–50.

The idea underlying the work described in this paper is that *T. congolense* inoculated into a young animal should give rise to a benign infection which confers immunity. It was found that when a calf 8 to 15 days old was inoculated with 2 to 5 cc. of the blood of a guinea-pig infected with *T. congolense*, the calf became infected, and after a few days exhibited numerous trypanosomes in the peripheral blood. When the infection was established the trypanosomes were found regularly in the blood; on certain days they were more numerous, and on others more scanty. After a period of 4 to 6 months, a stage was reached when trypanosomes were no longer found in the blood so regularly. Sometimes examination of the blood was positive and sometimes negative. The negative periods gradually lengthened, until, finally, a condition was reached when the trypanosomes could not be found, and the blood on inoculation no longer infected laboratory animals. Apparently, then, the animal had cured itself.

Re-inoculation of the cured calf with infected guineapig's blood failed to produce infection. The calf had hence acquired an immunity which was not due to a premunition, because trypanosomes no longer existed in the circulation. In a table details are given of 6 calves immunized in this way, and subsequently re-inoculated with infected guineapig's blood on 3 or 4 occasions. In only one of the calves on a single occasion was a single trypanosome found. This was apparently a temporary infection, because trypanosomes were never found again, and subsequent inoculation of virulent blood failed to produce infection. *W. Y.*

KLIGLER (I. J.) & BERMAN (M.). **Susceptibility and Resistance to a Trypanosome Infection. X.—Specific Character of the Immunity produced in Rats by the Injection of Suspensions of Dead Trypanosomes.**—*Ann. Trop. Med. & Parasit.* 1935. Dec. 18. Vol. 29. No. 4. pp. 457–461.

In a former paper Kligler & COMAROFF [this *Bulletin*, 1935, Vol. 32, p. 715] showed that repeated injection of a suspension of dead trypanosomes into rats enhances their resistance to an infection with the homologous strain of the organism. The present work was undertaken to determine whether this acquired resistance is specific.

The general technique was the same as that described in the earlier paper. A heavy suspension of trypanosomes in saline was incubated at 37°C. for 2 hours, and then left in the ice-box until all the trypanosomes were dead. This saline suspension of dead trypanosomes was used for immunization; each rat received 5 injections of 1·0 cc. on 5 successive days. The infecting dose was given 3 to 5 days after the last injection. Five experiments were carried out. In two the rats were treated with *T. evansi* suspension, and then equal groups were infected with *T. evansi*, *T. gambiense* and *T. equi*, respectively. In two the rats were treated with *T. gambiense*, and then groups were infected with the three species; in the last experiment the rats were treated with *T. equi* before injection with the three species of trypanosomes.

The results of the experiments are summarized in tables from which it appears that rats treated with suspensions of dead trypanosomes develop an increased resistance to an infection with the homologous strain. The enhanced resistance is specific; an infection with another species follows the same course as in control non-treated animals.

W. Y.

CORSON (J. F.). **Antelopes as Reservoirs of** *Trypanosoma gambiense.* [Correspondence.]—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. Apr. 8. Vol. 29. No. 6. p. 690.

Referring to the well-known experiments of BRUCE (1910) with *T. gambiense* and antelopes, Corson questions whether the existence of a chronic infection of the antelope with *T. brucei* was excluded. There seems little doubt that some at least of the antelopes became infected with *T. gambiense*, but whether the trypanosomes which were transmitted from the antelopes several months afterwards were *T. gambiense* or *T. brucei* appears to be doubtful, as no experiments were made on human beings. The importance of the subject makes it desirable that the experiments be repeated under stricter conditions.

W. Y.

BOURGUIGNON. Contribution à l'étude des trypanosomiases des suidés au Congo Belge. [**The Trypanosomes of Pigs in the Belgian Congo.**]—*Ann. Soc. Belge de Méd. Trop.* 1935. Dec. 31. Vol. 15. No. 4. pp. 491–499.

The observations upon which this paper is based were made during two virulent epidemics of trypanosomiasis in pigs, the first during the beginning of 1932 near Stanleyville and the second some distance north-west of Elizabethville.

The mild trypanosomiasis of pigs is mainly due to *T. congolense*. *T. brucei* is not found in pigs in nature, and these animals are immune to *T. vivax*. In 1921 WALRAVENS described a new species of trypanosome under the name *T. rodhaini*, which causes a virulent disease of pigs in the vicinity of Elizabethville. Bourguignon comes to the conclusion that *T. rodhaini* is only a variety of *T. simiae*. He then passes to a consideration of the virulent trypanosomes of pigs described by SCHWETZ (1930) and also of those found by himself in the Province Orientale in 1932 and by himself and JUSSIANT at Katanga.

The general conclusion is that the chief characters of the trypanosomiasis of pigs are firstly its great virulence for the pig and the impossibility of transmission to the usual laboratory animals, and secondly a morphology which is predominantly that of *T. simiae*. He points out that *T. simiae* resembles *T. gambiense* in that it is made up of two extreme forms, the one without and the other with a free flagellum and intermediate forms. *W. Y.*

QUARTERLY BULLETIN OF THE HEALTH ORGANISATION. LEAGUE OF NATIONS. Geneva. 1936. Mar. Vol. 5. No. 1. pp. 168–181. **Animal Diseases communicable to Man. C. Trypanosomiasis.** [Report of Committee on Diseases conveyed by Animals to Man and Resolutions of Pan-African Health Conference, Johannesburg, Nov. 20–30, 1935. pp. 168–169.] DU TOIT (P. J.). **Trypanosomiasis of Domestic Animals.** [Annex I. pp. 170–179.] MACLEAN. **Notes on Trypanosomiasis.** [Annex II. pp. 179–181.]

These articles are of a general nature and contain nothing new.

W. Y.

BOURGUIGNON (G. C.) & VAN DEN BERGHE (L.) with the collaboration of Ludo VAN BOGAERT. La trypanosomiase expérimentale du cynocéphale par voie intrarachidienne. [**Experimental Trypanosomiasis of the Baboon after Intrathecal Inoculation.**]—*Ann. Soc. Belge de Méd. Trop.* 1936. Mar. 31. Vol. 16. No. 1. pp. 9–36. With 12 figs. on 6 plates.

Information is given of attempts to infect *Papio jubilaeus* intrathecally with *T. brucei* and *T. gambiense*.

Five animals were given 2 or 3 lumbar injections of the blood of guineapigs heavily infected with a strain of *T. brucei* which had been passaged through 40 guineapigs ; and six animals received 2 injections, at a month's interval, of the blood of a guineapig infected with a strain of *T. gambiense* from a Mayumbe (Congo Belge) patient, which had been passaged through 22 or 23 guineapigs. Details are given of each of the 11 inoculated baboons.

Of the 5 animals inoculated with *T. brucei* one died a year later and another 2 years later ; examination of the brains furnished no evidence of trypanosomiasis. A third baboon remained negative for a year and then died, but no post-mortem was made ; the other two remained healthy for 3 years.

Of the 6 animals inoculated intrathecally with *T. gambiense*, two died after 2 years without signs of trypanosomiasis, one was accidentally killed after 2 years and was apparently healthy, and two others remained well for 3 years. Only one animal died 2 years after inoculation

from characteristic cerebrospinal trypanosomiasis. The course of the infection was very interesting. The baboon did not exhibit any symptoms, and its cerebrospinal fluid remained but slightly changed (30 cells per mm. with 0·60 of protein and free from trypanosomes) for almost two years; suddenly at the beginning of the third year it was discovered that the cerebrospinal fluid contained more than 200 cells per cmm. and trypanosomes, which, on inoculation into guineapigs and mice, proved to be *T. gambiense*. Three days later the monkey exhibited very definite symptoms of cerebrospinal infection and died of the disease about four months later.

A study of the pathology of the brain was made by van Bogaert, and his findings have already been summarized in a previous paper [see above p. 657].

The next portion of the paper deals with experiments undertaken to ascertain whether the serum and cerebrospinal fluid of *Papio jubilaeus* of Katanga exhibited the protective or curative action commonly seen in the serum of baboons. The fluids were tested on guineapigs infected with *T. gambiense*, and the results failed to provide any evidence that either the serum or the cerebrospinal fluid of *Papio jubilaeus* exerted a protective or curative action.

In view of this, it was decided to inoculate two young *Papio jubilaeus* intraperitoneally, with a view to ascertaining whether they could be infected in this way. In the first the trypanosomes persisted in the peripheral circulation in small numbers for some days. The second, however, showed a definite blood infection. About two weeks after inoculation numerous trypanosomes were found in the peripheral blood and the animal had a definite febrile disturbance. In a day or two the trypanosomes disappeared from the blood, which remained negative, apart from one occasion when very scanty parasites were found, until the death of the animal, which took place two months after inoculation. Before death the baboon developed palsy in its posterior limbs, but its blood was negative and post-mortem examination failed to reveal any lesion which could be definitely ascribed to trypanosomiasis.

The authors are of opinion that this blood infection of *Papio jubilaeus* in Katanga confirms their previous experimental findings that the serum does not contain protective substance. *W. Y.*

Herrick (C. A.) & Cross (S. X.). **The Development of Natural and Artificial Resistance of Young Rats to the Pathogenic Effects of the Parasite** *Trypanosoma lewisi.*—*Jl. Parasitology.* 1936. Apr. Vol. 22. No. 2. pp. 126–129.

A wild strain of *T. lewisi* was found to be pathogenic to young and not to old rats. Its virulence did not vary to any appreciable extent; it never became pathogenic to old rats and remained pathogenic to young ones. Another strain of *T. lewisi*, which had been maintained in laboratory animals for 7 years, behaved in exactly the same way.

The authors set themselves the problem of determining the age at which young rats became resistant to the pathogenic effects of this parasite. White rats in good condition were employed. The test animals were separated into groups of various ages ranging from 2 to 40 days. In every case, except in the two-day-old group, the infected and control animals were all male litter mates. The inoculations were

made intraperitoneally, and the number of trypanosomes given varied according to the weight of the animal.

The rats were divided into two categories, (1) those that were allowed to suckle their mothers and (2) those that were weaned. The suckling young were divided into 4 groups according to age, as is shown in the following table :—

TABLE I.

Showing the death rate in a group of rats infected with T. lewisi *and their uninfected litter-mate controls.*

	Suckling				Weaned		
	Experimental						
Age in days	2	10	20	30	20	30	40
No. of rats	10	20	50	25	118	25	25
No. of deaths	0	0	2	0	100	19	0
Percentage which died ...	0	0	4	0	84·7	76	0
	Uninfected controls						
Age in days	2	10	20	30	20	30	40
No. of rats	0	20	43	0	98	25	0
No. of deaths	0	0	0	0	3	1	0
Percentage which died ...	0	0	0	0	3·1	4	0

From these results it appears that there is a very definite age resistance developed by the young rats to the pathogenic effects of *T. lewisi*, and that the resistance is well established by the time the rats are 40 days old. It is also apparent that for some reason, which cannot be at present explained, the suckling rats were protected from the effects of the parasite. They were not immune to infection, because all became infected and showed a large number of trypanosomes in their peripheral blood. Furthermore, the mothers were shown not to be immune, as they were successfully infected at the close of their lactation period.

Owing to the fact that the development of age immunity was so definite, an attempt was made artificially to increase the resistance of the young rats so they would not be affected by trypanosomes. This was done successfully in two ways: (1) by implanting the pituitary glands of adult non-immune rats intramuscularly into the young susceptible animals, and (2) by injecting oestrin subcutaneously into the young rats. Why the implanting of pituitaries or the injection of oestrin made them more resistant to the effects of an infection of *T. lewisi* cannot be answered at this time, but work on the problem is proceeding. *W. Y.*

REINER (L.), SMYTHE (C. V.) & PEDLOW (J. T.). **On the Glucose Metabolism of Trypanosomes** (*Trypanosoma equiperdum* **and** *Trypanosoma lewisi*).—*Jl. Biol. Chem.* 1936. Feb. Vol. 113. No. 1. pp. 75–88. [14 refs.]

This paper reports experiments by which the main steps in the breakdown of glucose by *Trypanosoma equiperdum* and *T. lewisi* have

been established; it is of a technical nature and must be consulted in the original by those interested. Apparently the mechanism of glucose decomposition is very different in the two cases; while *T. equiperdum* produces two 3-carbon atom compounds, *T. lewisi* forms a 2- and a 4-carbon atom. *W. Y.*

TALIAFERRO (William H.) & PAVLINOVA (Yelena). **The Course of Infection of *Trypanosoma duttoni* in Normal and in Splenectomized and Blockaded Mice.**—*Jl. Parasitology.* 1936. Feb. Vol. 22. No. 1. pp. 29–41. With 5 figs. [11 refs.]

The authors summarize their paper as follows:—

"1. The normal course of infection of *T. duttoni* in the normal white mouse approximates that of *T. lewisi* in the normal white rat, but there are striking quantitative differences:

"(*a*) Reproductive activity in *T. duttoni* is less and generally only a fraction of that observed in *T. lewisi* during the first of an infection, but continues at a minimal rate for several weeks.

"(*b*) The number of trypanosomes is ordinarily much less than in infections of *T. lewisi*.

"2. Both the reproductive activity of *T. duttoni* and the severity of resulting infections are greatly enhanced by splenectomy and India ink blockade carried out prior to infection. They are also adversely affected by intercurrent infections. Although part of this difference is due to a lowering of acquired resistance (ablastin formation), it is largely due to a lowering of the natural resistance of the mouse. This fact is of particular interest in showing that both natural and acquired resistance to the reproduction of the trypanosomes is dependent upon macrophage function."

W. Y.

RODHAIN (J.) & HENRY (E.). Localisation cutanée du *Trypanosoma marocanum* chez le cobaye. [**Cutaneous Localization of *Trypanosoma marocanum* in the Guineapig.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 13. pp. 1388–1390. With 2 figs.

In a previous paper Rodhain has drawn attention to cutaneous lesions which appeared in a guineapig infected with *T. marocanum*.

In general, guineapigs infected with the authors' strain of *T. marocanum* succumbed from the acute disease within about 30 days. Sometimes towards the end of the infection the external genitalia and neighbouring regions became oedematous. Whilst the dermis itself was thickened, it was especially the infiltration of the sub-dermal tissues which characterized this external manifestation of the acute infection.

With the object of obtaining chronic infections, guineapigs were treated with subcurative doses of fouadin. It was found that 0·2 or 0·3 cc. of neo-antimosan given subcutaneously caused the disappearance of trypanosomes from the peripheral blood for 10 to 15 days. If the treatment was repeated when the animals relapsed it was sometimes possible to keep them alive for several months, although in order to do so particular care was necessary, especially when the animals developed pronounced oedema under the abdomen.

By this means it was possible to follow closely the slow development of the infection; and the presence of trypanosomes in the blood and in the skin was observed systematically at various stages of the disease. The blood from the ear was examined in thick drop preparations.

In order to obtain a drop of serous fluid from the skin a nick was made with a fine pair of scissors and pressure applied to the surrounding oedematous region by the thumb and forefinger: in this way a drop of serous fluid containing only a few red cells was caused to exude. It was observed that in these chronic infections trypanosomes appeared in the skin after one or two trypanolytic crises. They were often numerous in the skin when scanty in the blood. [A similar state of affairs is, of course, well known in rabbits and other animals in which trypanosomiasis is a chronic infection.] *W. Y.*

PANNIER (R.). Essai de culture de *Trypanosoma lewisi* dans le latex d'*Euphorbia characias* et *Euphorbia esula*. [**Attempt at culturing** *T. lewisi* **in the Latex of** *Euphorbia characias* **and** *Euphorbia esula*.] —*C. R. Soc. Biol.* 1936. Vol. 122. No. 16. pp. 29–30.

In 1923, FRANCHINI attempted to cultivate different flagellates in latex, but he obtained only moderate success. The author has tried to adapt *T. lewisi* to a latex. In his first experiments he used a mixture consisting of equal parts of blood and latex, and cultured the trypanosomes in hanging drop preparations. After 60 hours [temperature not stated] the trypanosomes were immobile and would not stain well. As the controls, in which blood alone was used, gave similar results, it is concluded that the latex was not toxic to the trypanosomes.

In later experiments the cultures were made in tubes. The latex of *Euphorbia characias* was mixed in different proportions with the semifluid medium of Wenyon, and the mixture sterilized at 120°C. for 15 minutes. Under such conditions living trypanosomes, which stained well, could be found in certain tubes up to the 5th day. The following day the trypanosomes seemed to have lost their undulating membrane and there were numerous agglutinations.

The author believes that experiments of this sort will establish a parallelism between the flagellates of Euphorbia and those of insects and of vertebrates. *W. Y.*

SINGER (Ernst). Die Wirkung der Chemotherapeutica auf die Trypanosomenzelle. [**The Action of Chemotherapeutic Substances in Trypanosome Cells.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1936. Feb. 10. Vol. 117. No. 6. pp. 752–756.

Experiments were undertaken with the object of investigating, by means of the methylene blue decolourization method of Thunberg, the influence of chemotherapeutic substances on the dehydrases of trypanosome cells.

The technique employed was briefly as follows:—

In vitro experiments: A suspension of trypanosomes was obtained in plasma and sufficient 5 per cent. glucose and 0·1 per cent. methylene blue solutions added to make the final concentration of glucose 0·5 per cent. and that of methylene blue 1 in 50,000. To each of a series of small tubes 0·9 cc. of this suspension was added, and then 0·1 cc. of suitable concentrations of various drugs. After the air in the tubes had been exhausted by a powerful water pump the tubes were sealed and placed in a water bath at 37°C.

In vivo experiments: Mice at the height of a nagana infection were given an intramuscular injection of the various drugs, and then bled after suitable intervals. Trypanosome suspensions containing about 100 million trypanosomes per cc. were prepared and dealt with as above.

The action of the various drugs on the discolourization time of the methylene blue due to hydration of the trypanosomes was then examined. The results are given in a table in which the values are based on the time taken for the control tubes (without drug) to become decolourized. As the number of trypanosomes varied in the different experiments, the figures in the table which represent the results obtained in a number of experiments are calculated on the assumption that the controls were discoloured in 100 units of time.

The table shows that all the drugs which act on trypanosome infections, *e.g.* atoxyl, solusalvarsan, sulfoharnstoff, germanin and trypaflavin, hinder dehydration when present in higher concentrations, and increase it when present in lower concentrations.

From the *in vivo* experiments it appears that the trypanosomes, removed 20 to 30 minutes after the injection of an active dose of a chemotherapeutic substance, show a retardation of the dehydration process.

The injection of active doses of a chemotherapeutic substance causes the disappearance of trypanosomes from the circulation through phagocytosis by the cells of the reticulo-endothelial system. It is known that this system takes up only such substances as wander towards the anode. Normally, trypanosomes migrate towards the kathode, and the question arises whether chemotherapeutic substances so change trypanosomes that they tend to pass towards the anode.

Eosin is not taken up by normal trypanosomes. When, however, this dye is injected into an infected mouse 2 to 4 hours after a dose of germanin, sulfoharnstoff or neosalvarsan, it is found, when blood preparations are examined under the fluorescence microscope, that scanty trypanosomes have taken up the dye. The reason why so few stained trypanosomes are to be found is that the damaged individuals are immediately removed from the circulation by the reticulo-endothelium. *W. Y.*

GRUHZIT (O. M.) with the assistance of W. D. LINDSAY, G. HENDRICKS & M. C. DODD. **Mapharsen ("Arsenoxide") in the Therapy of Experimental Syphilis and Trypanosomiasis.**—*Arch. Dermat. & Syph.* 1935. Dec. Vol. 32. No. 6. pp. 848–867. With 7 figs. [18 refs.]

Experiments described in this paper were undertaken with the object of ascertaining the value of arsenoxide in the treatment of spirochaetosis and trypanosomiasis. The preparation used is called "mapharsen"; it is meta-amino-para-hydroxyphenylarsine oxide and contains 29·01 per cent. of metallic arsenic.

The M.L.D. of mapharsen for albino rats given intravenously was about 20 mgm. per kilogram and the M.T.D. about 18 mgm. For rabbits the M.L.D. used was about 16 mgm. and the M.T.D. about 14 mgm. per kilogram. When given intramuscularly the M.L.D. for rats was 24 mgm. per kilo., and the M.T.D. about 22 mgm.

When injected intravenously into dogs in a dose of 1 mgm. per kilo. twice weekly for 9 doses, and in a dose of 2 mgm. twice weekly for six doses, the drug caused neither albuminuria nor an increase in the total nonprotein nitrogen or the sugar content of the blood. The administration was not followed by any untoward symptoms, such

as diarrhoea, nausea, vomiting or general weakness. A dose of 3 mgm. per kilo. caused occasional salivation, and a dose of from 4 to 5 mgm. once weekly frequently caused vomiting, salivation and albuminuria. Intramuscular injections into dogs of 5 mgm. per kilo. once weekly for 8 weeks caused neither vomiting nor albuminuria.

The M.E.D. of mapharsen given intravenously to albino rats infected with *T. equiperdum* was 1 mgm. per kilo. as compared with 10 mgm. and 22 mgm. for arsphenamine and neoarsphenamine, respectively. The therapeutic indices of the three substances were 18, 14, and 9, respectively.

The sterilizing doses for rats infected with *T. equiperdum* were 2 mgm., 12 mgm. and 24 mgm. per kilo. for mapharsen, arsphenamine and neoarsphenamine, respectively; and the corresponding curative indices were accordingly 9, 11·6 and 8·3, respectively.

In rabbits 1 mgm. per kilo. of mapharsen cleared lesions (the oedema subsided and the lesions became dry and cicatrized) due to *Sp. pallida* in from 12 to 36 hours. A dose of 10 mgm. per kilo. of neoarsphenamine cleared similar lesions in from 24 to 48 hours. The lesions in syphilitic rabbits had healed completely within 7 days after doses of 1 mgm. and 10 mgm. of mapharsen and neoarsphenamine, respectively.

The protective effectiveness of mapharsen in doses of 1 mgm. per kilo., given 48 hours after inoculation with *Sp. pallida* and repeated three times in two weeks, or in a single dose of 2 mgm., was about 100 per cent. for rabbits: a single dose of 30 mgm. of neoarsphenamine was required to protect 100 per cent. of the rabbits. With single doses the sterilizing indices of the two drugs were about equal, but with repeated doses mapharsen appeared superior to neoarsphenamine. Intramuscular injections of mapharsen possessed about the same therapeutic value as intravenous injections.

The general conclusion reached is that mapharsen in experimental trypanosomiasis and syphilis appears to be well tolerated, and to possess at least as high a therapeutic value as neoarsphenamine.

W. Y.

ROUBAUD (E.) & TREILLARD (M.). Infection expérimentale de *Glossina palpalis* par un coccobacille pathogène pour les muscides. [**Experimental Infection of** *G. palpalis* **by a Coccobacillus Pathogenic for Muscidae.**]—*Bull. Soc. Path. Exot.* 1936. Feb. 12. Vol. 29. No. 2. pp. 145–147.

Recently the authors have described the spontaneous infection of *G. morsitans* by a coccobacillus. This is the first pathogenic organism for tsetses known. It can be isolated in pure cultures, and from the cultures fresh *G. morsitans* can be infected. The infection is taken up by the proboscis when the glossina bites an animal the skin of which is soiled by the culture. The *G. morsitans* dies of a septicaemia within one to three days after biting the skin of a guineapig soiled with a 24-hour culture of the organism. The coccobacillus is equally pathogenic for *Musca domestica.*

The present paper records experiments to ascertain whether the organism is pathogenic for *G. palpalis.* It was found that this tsetse did develop an infection of the coccobacillus, but that it was less sensitive to the infection than was *G. morsitans.* *W. Y.*

CHAGAS (Evandro). Summula dos conhecimentos actuaes sobre a Trypanosomiasis americana. [**Review of Present Knowledge of American Trypanosomiasis.**]—*Mem. Inst. Oswaldo Cruz.* 1935. Vol. 30. No. 3. pp. 387–416.

As the title indicates this article summarizes the present knowledge of the parasite, *T. cruzi*, its characters, transmission, reservoir hosts, the symptoms it produces and the pathology of the infection. No one could do this better than Dr. Evandro Chagas, who has been occupied in close study of the disease since it was first described by Carlos CHAGAS in 1909. The author reviews in order the geographical distribution and incidence of the disease, the Trypanosome and its evolution in the insect transmitter and in the vertebrate hosts. He considers the question of *T. verpertilionis* in bats, its similarity to *T. cruzi*, but suspends judgment as to its identity with the latter. The symptoms of the disease in man and the pathology are described. The former according to whether the form is acute or chronic, and cardiac or nervous. That the disease occurs in goitrous districts is known but the older idea that there was a goitrous form of the disease is no longer maintained ; as the author states " Up to the present there is no experimental confirmation of [Carlos] Chagas' theory " that the goitre occurring in these regions is due to infection by *T. cruzi*. There follows a short section on diagnosis and a few words on prophylaxis. " Specific treatment at present there is none. Drugs with trypanocidal action have been tried by many investigators without success." (Não existe, até ao presente momento, tratamento especifico para a trypanosomiase americana. Medicamentos de acção trypanosomicida teem sido experimentados por numerosos pesquizadores sem qualquer exito.)

H. H. S.

JOHNSON (C. M.) & DERIVAS (G. T.). **Six New Cases of Chagas' Disease in Panama with Review of Previous Cases.**—*Amer. Jl. Trop. Med.* 1936. Jan. Vol. 16. No. 1. pp. 47–57.

Since the discovery of Chagas' disease in Panama in 1931, 13 cases have been reported, one of which terminated fatally. The authors give details of this additional case, including two fatalities collected by them since March, 1935.

Apparently no age is exempt from the disease, as the youngest patient was three months and the oldest 74 years of age. Seven of the 19 cases occurred in children under three years of age, and of these three ended fatally. Of the non-fatal cases all but two were discovered accidentally during malarial surveys. In this group symptoms and signs of Chagas' disease were practically absent. There was a history of general malaise accompanied by a few paroxysms of fever.

In the three fatal cases reported in this paper, as well as in the fatal case reported by DECOURSEY, the outstanding lesion in the heart was a diffuse inflammatory type associated with a considerable degree of damage to the muscle fibres themselves. In the brain the lesions were focal and consisted of collections of nests of cells in which occasionally were seen clumps of parasites. The neuroglia were the elements which were infected. In both the heart and brain the various developmental stages of the parasites were seen.

In view of the scarcity of the trypanosomes in the peripheral blood diagnosis is a matter of difficulty, and it would seem not unlikely that

in the past some cases of Chagas' disease have been mistaken for malaria. The authors consider other reasons for believing that the incidence of Chagas' disease on the Isthmus of Panama is higher than has been suspected hitherto. *W. Y.*

MAZZA (Salvador), BENITEZ (Clemente) & JANZI (Edin Z.). Primer caso de forma aguda de enfermedad de Chagas y primeros animales domésticos portadores de *S. cruzi*, comprobados en Corrientes.—*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1936. Publicación No. 26. pp. 28–33. With 4 figs.

TORREALBA (J. F.). Consideraciones sobre la enfermedad de Chagas en Zaraza.—*Gac. Méd. de Caracas.* 1935. Dec. 15. Vol. 42. No. 23. pp. 356–361. With 4 figs. (1 map). [41 refs.]

MAZZA (Salvador). Infestación de *Triatoma* por *Schizotrypanum cruzi* y hallazgo de animales domésticos portadores del mismo en el sur de la República (Rio Negro y provincia de Buenos Aires). Nueva especie de *Triatoma* determinada en Rio Negro. [**Triatoma Infestation by** *T. cruzi*; **Domestic Animals as Carriers in the Argentine. A New Species of Triatoma discovered in Rio Negro.**] —*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1936. Publicación No. 26. pp. 1–19. With 12 figs. [12 refs.]

In the Rio Negro territory [and also elsewhere] *Triatoma infestans* exists in large numbers and heavily infected with *T. cruzi*. *Triatoma platensis* may be found in the same localities, in fact in the same dwellings, as *T. infestans*, but not parasitized. A new species is described in detail in the text, with illustrations, and a table of measurements of the different parts, comparing them with *T. platensis*, male and female, which it closely resembles in some respects. The new species is called *Triatoma rosenbuschi*, after Professor F. ROSENBUSCH who was the first to study these insects (vinchuca). Three species of Triatoma have now been found in the Argentine naturally infected, *T. infestans*, *T. oswaldoi* and *T. rosenbuschi*. In a house in the rural district of Fuerte General Roca a kitten was found carrying *T. cruzi*.

H. H. S.

CHAGAS (E.). Infection expérimentale de l'homme par le *Schizotrypanum cruzi*. [**Experimental Infection of Man by** *Schizotrypanum cruzi*.]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 8. pp. 769–771.

The author has continued his investigations on the experimental infection of the human being with *T. cruzi*. A volunteer (No. 7) was inoculated with the faeces of *Panstrongylus megistus* containing numerous crithidial and metacyclic forms of *T. cruzi* of human origin. The temperature rose 5 days later and trypanosomes were shown to be present in the blood by subinoculation into a guineapig on the 14th day, but were not found by direct examination by the 19th day. On the latter day the patient was re-inoculated with 2·5 cc. of the blood of a young dog containing numerous *T. cruzi*. The temperature, which had not risen beyond 38·5°C. the four previous days, rose the day after the second inoculation to 39·5°C. Six hours after the second

inoculation parasites (both narrow and broad forms) were found by direct examination of the blood. The number remained stationary for 3 days and then gradually decreased until by the 8th day only a few broad forms were to be seen.

This observation proves the existence of an initial phase of blood infection. The parasites seen 6 hours after inoculation could not have come from the tissues because the time did not suffice and because both broad and narrow forms were seen. The number of parasites inoculated on the first occasion was probably too few to enable them to be seen in blood examination, and possibly the metacyclic forms have a greater capacity for penetrating the tissues. The steady diminution in the number of trypanosomes seen in the blood during the days following the second inoculation was probably due to the trypanocidal action of the blood and to the localization of the remaining parasites in the tissues.

The organs of volunteer No. 6, who died from malignant disease 5 months after inoculation with *T. cruzi* from a *Tatusia novemcincta*, were examined. This patient also had exhibited an initial acute infection with parasites in the blood and a certain amount of fever. No parasites were found in the organs usually involved, but sections of the liver showed numerous foci of infiltration by lymphocytes, plasma cells and macrophages. *W. Y.*

KOFOID (C. A.), WOOD (F. D.) & MCNEIL (E.). **The Cycle of *Trypanosoma cruzi* in Tissue Culture of Embryonic Heart Muscle.**—*Univ. California Public. Zool.* 1935. Vol. 41. No. 3. pp. 23–24.

Details are given of the cyclic growth of *Trypanosoma cruzi* in tissue cultures of embryonic heart of rat and mouse.

The tissue cultures were made from 14- to 16-day rat and mouse embryos. They were grown in plasma-embryo extract medium on 7·4 × 4·5 cm. slides having a depression 4 mm. deep. They were incubated 24 hours at 37°C. before being inoculated with culture forms in semi-solid blood agar medium of the California strain of *T. cruzi* from the wood rat. Cultures were examined at frequent intervals during the first 24 hours after inoculation, and at least twice daily thereafter for 6 days.

Some of the crithidial culture forms used in the inoculum survived in the medium as long as the experiment lasted (7 days) and apparently continued to multiply. During the first 24 hours there was a decrease in the number of trypanosome forms in the culture medium, these seemingly being the forms which entered the tissues. After 48 hours the free-swimming trypanosomes were very rare or absent. In cultures 24 hours old trypanosomes and many crithidias could be seen adhering to the muscle tissue, fibroblasts, and macrophages. Although several of the adherent forms were watched for 20 minutes actual penetration of the cells was not observed.

The opacity of the muscle made it difficult to determine what was taking place within it, but the macrophages, fibroblasts, and extracellular parasites could be easily observed. At 24 hours many macrophages contained from one to several motile flagellates. On the third day granular débris from degenerating macrophages was observed to contain wriggling flagellates. No flagellates were seen in the fibroblasts.

Trypanosomes of the type found in the circulating blood were first seen on the 5th day after inoculation. They were at first localized

as they emerged from the massed leishmaniform phases, and were generally shorter than the long slender metacyclic forms which occur in the culture tube and insect vector. These blood forms were often found in loosely associated groups in the plasma surrounding the heart muscle from which they had recently emerged.

In the sectioned material of 24-hour preparations, macrophages containing one or two parasites were numerous. These parasites were either in the crithidia or leishmania stage, probably the former. Although these crithidial stages are not supposed to be infective, it is obvious that they enter or are ingested by macrophages, and that they may survive the death of the cell. Possibly, they eventually develop into infective forms, either within or outside of body cells.

Groups of typical leishmaniform bodies were first seen in sectioned heart muscle on the 4th day after inoculation. Some of the groups were typical with no flagella, whilst others were of the stubby crithidial type with short flagella. A few groups of very slender trypanosomes with round or oval parabasals were noted.

The authors conclude that the development of *T. cruzi* in tissue cultures of heart muscle is the same, with respect to time and stages of its cycle, as its development in the body of the living vertebrate host. *W. Y.*

MAZZA (Salvador) & PALAMEDI (Benito). Forma aguda benigna de enfermedad de Chagas observada en Barranqueras, Chaco.—*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1936. Publicación No. 26. pp. 19–22. With 1 fig.

MAZZA (Salvador) & VALLE (Fernando A.). Forma aguda benigna de enfermedad de Chagas observada en Puerto Tirol, Chaco.—*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1936. Publicación No. 26. pp. 25–28. With 1 fig.

MAZZA (Salvador) & CORSI (Eduardo M.). Tercera observación de forma aguda benigna de enfermedad de Chagas en Presidente de la Plaza, Chaco.—*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1936. Publicación No. 26. pp. 22–25. With 2 figs.

MAZZA (Salvador) & MAINOLI (Martin R.). Forma aguda benigna de enfermedad de Chagas comprobada en el departamento de Anta (Prov. de Salta).—*Universidad Buenos Aires: Misión de Estudios de Patología Regional Argentina Jujuy.* 1936. Publicación No. 26. pp. 34–39. With 1 fig.

CUENCA (Heberto). La forma cardiaca de la enfermedad de Chagas, con presentación de nuestro primer caso.—*Gac. Méd. de Caracas.* 1935. Dec. 15. Vol. 42. No. 23. pp. 361–365. With 3 figs.

MALARIA.

PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE. 1936. Mar. Vol. 29. No. 5. pp. 537–562. (Sect. of Epidemiol. with Trop. Dis. pp. 1–26). With 19 figs.—**Discussion on the Malaria Epidemic in Ceylon 1934–35** [BRIERCLIFFE (R.), DALRYMPLE-CHAMPNEYS (Weldon), WIGGLESWORTH (V. B.) & others].

This discussion was of great general interest; it comprised:—Examples of misleading reports in the lay press. An outline of the measures taken and the difficulties of the task. The opener's view of the main factor in the causation of the epidemic. The sudden onset of the epidemic. The predominance of benign tertian. The secondary wave. Remarks by Dr. Wigglesworth on the habits of *A. culicifacies*. Col. James' theory of the action of susceptibility as a factor in the epidemic. Professor Schulemann's remarks on exaggerated claims for atebrin musonate. His standard treatment. Dr. Kikuth's opinion of the danger of intravenous injections. The discussion was opened by Sir Weldon Dalrymple-Champneys who read a joint paper prepared by Dr. R. Briercliffe and himself.

Misleading reports. Difficulty of task. Measures taken.—Very misleading accounts of the epidemic appeared in the lay press; for instance a report of 5,000 deaths in a particular district became 50,000 in an English newspaper, and had swollen to 500,000 by the time it reached America. Again, a reporter cabled that there was a "green caterpillar pest" and that thousands were being buried in the Kurunegalla district. In the American papers, the word "catapillar" was omitted, it was reported that thousands were dying from the green pest and numerous requests were received by the Ceylon medical department for specimens of the "green malarial parasite." The Opener said he could testify as an eye-witness to the splendid way in which Dr. Briercliffe had handled this unprecedented epidemic in very difficult circumstances and in the face of much hostile criticism and misrepresentation. It had been suggested by critics that the epidemic could have been brought to an earlier conclusion by widespread antilarval measures. Carefully drawn maps and calculations show that it would have been necessary to treat about 7,000 miles of watercourses. A coolie employed in antilarval work deals with about 1½ miles a day at a cost of about 10 rupees a mile. The cost would have been out of proportion to the benefit to be expected, even if the trained personnel necessary for such work had been available, which was not the case. Dr. V. B. Wigglesworth who contributed the second paper of this Discussion, stated "it was noted in Ceylon during the epidemic that *culicifacies* larvae occurred not only in the marginal pools, but also in the main bodies of water in the rivers. That means that the larvae must be carried downstream, and however well one stretch of river is controlled, fresh larvae will be continually brought downstream to it." He said that the three spots which he visited in the East where he felt least hopeful about antimosquito measures in rural areas were the three places—the Punjab, the Madras Agency, and Ceylon—where *culicifacies* was the chief malaria carrier. In spite of all these difficulties a mobile organization, consisting of locally recruited labour under trained personnel was rapidly built up in Ceylon and some 300 miles of the four main rivers and their tributaries were brought under control, oiling being confined to areas in

the neighbourhood of large villages. "The most important anti-malarial measure undertaken was the treatment of patients suffering from the disease. The standard treatment for adults was 7½ grains of quinine sulphate or bisulphate in solution three times a day." Additional measures undertaken to cope with the outbreak have been described already in this *Bulletin* (see Briercliffe and also James, above).

Main factor in causation of epidemic.—The authors of the opening paper consider that the main factor in the causation of the epidemic was the prolific breeding of *A. culicifacies* in pools left in the beds of the large rivers which had been dried up by the drought. Similar pools are often present in smaller rivers, even in normal years, but, because the narrow streams are shaded, *A. culicifacies* does not breed in them; it selects shallow pools of still, clear water exposed to sunlight. The reason that Galle and Matara, in the extreme south of the wet zone, remained free from the epidemic was because the rainfall for the year in these districts was up to the average, no actual drought occurred, the rivers were flowing as usual, *culicifacies* was not found and malaria did not occur.

Did the epidemic begin suddenly?—There is some difference of opinion as to the manner in which the epidemic began; the authors state that it began suddenly and they write, "The explosive nature of the onset was at first the characteristic feature of the epidemic. . . . The epidemic . . . burst with amazing suddenness in the Deduru Oya basin, which in the course of a week was completely involved."

Type of parasite and infection rate of anopheles.—Throughout the epidemic benign tertian infections predominated, the relative proportions of the three parasites from November 1934 to March 1935 being *P. vivax* 62·2, *P. falciparum* 36·7, and *P. malariae* 1·1 per cent. The proportion of subtertian infections increased, however, from 24·7 in November to 43·4 in January, and fell to 28 per cent. in March. In December, 12·9 per cent. of the *culicifacies* were infected and in some places the infection rate was as high as 19 per cent. Out of 5,063 anopheles collected in dwellings from November onwards, 88·5 per cent. were *A. culicifacies.*

Was the secondary wave due to recurrences?—"At the end of the first week of April, a secondary wave of malaria started to sweep over the epidemic area. It was not unexpected. . . . The disease generally was of a much milder character during the secondary wave than during the early months of the epidemic and deaths were not so numerous." This secondary wave was not due only to recurrences, because "many persons who had not previously suffered from malaria were infected, certain villages which had hitherto escaped lightly or even completely were involved, and in the Mahaweli Ganga basin new areas were invaded. But the most interesting extension of the epidemic was upwards, . . . in May and June, malaria broke out in villages on the banks of streams situated 3,500 to 4,000 feet above sea-level. . . . Previously at these elevations, the larvae of *culicifacies* had rarely been found and the adults never."

Dr. V. B. Wigglesworth spoke of the habits of *A. culicifacies* in different parts of India, (*a*) In Assam, it breeds in myriads during the dry season in clear pools left in the river beds; but it is of no importance because this is the cold season and no transmission takes

place. Here *A. minimus* breeding in the grassy-edged streams is the carrier. (*b*) In the Anamalai Hills of South India, *A. culicifacies* breeds in the rivers and streams, but it does not thrive there during the season suitable for transmission (March to June) and it is of no importance. Here *A. fluviatilis*, a close ally of *varuna*, is the carrier. (*c*) In the Punjab *A. culicifacies* finds breeding places every year in the smaller irrigation canals, in seepages from the larger canals, and in pools in dried up water-courses. Villages close to these places are centres of endemic malaria. These places have much in common with the malarious dry zone of Ceylon. In years of exceptional rainfall innumerable temporary breeding places are created, wide tracts of the Punjab become infested with *A. culicifacies* and, if these conditions develop after an interval during which the people have lost their immunity to malaria, devastating epidemics result. As in Ceylon, an epidemic is determined by the coincidence of an abundance of *culicifacies* and a non-immune population. Excessive rainfall favours *culicifacies* in the Punjab, excessive drought in south-west Ceylon. In another part of India, however, the hilly districts of the Madras Agency, the malaria season begins when the rivers dry up and contain residual pools. Here, it is not an exceptional phenomenon, as in Ceylon, but an annual event in a region of hyperendemic malaria.

Colonel S. P. James, enunciated a theory in explanation of the importance of lack of immunity in determining an epidemic (see also James below, p. 684). Why is lack of immunity an important factor in causing an epidemic? "According to the results of laboratory work at Horton the answer was that in non-immune persons suffering from malaria, gametocytes appeared in the blood more frequently and in far greater numbers than in immune persons. . . . When anopheles fed upon these patients their stomachs became crowded with from 300 to 800 oöcysts, with the result that enough sporozoites were produced to infect many people and to keep the insect infective throughout its life. But when anopheles fed upon malaria patients possessing some immunity, the number of oöcysts found on their stomachs seldom exceeded 10, with the result that the sporozoites produced were all used up in biting two or three people and the mosquito remained infective for only a very few days. . . . Lack of immunity in the population at risk was the first requirement for the production of heavily infected mosquitoes and therefore it was an important factor in the causation of an epidemic."

Professor W. Schulemann said that Simeons had stated that in peroral treatment with atebrin, three days must be lost until the liver became saturated with the drug, but that this statement was not in accordance with facts. He considered that Simeons' practical conclusions were too far-fetched. A "*therapia sterilisans magna*" by injecting large doses was impracticable, peroral treatment was to be preferred except in very serious cases, and should be specially chosen to prevent relapses. He thought that atebrin musonate had many advantages over quinine when injections were required. The standard treatment to be recommended was :—Atebrin 0·3 gram daily by the mouth for 5 to 7 days; then, plasmoquine 0·02 gram daily for 3 or 4 days. Subsequently 0·2 gram atebrin and 0·02 gram plasmoquine on one day of the week, and 0·2 gram atebrin alone on another day of the week. But where good supervision was not possible, or where there was a floating population, atebrin should be given without plasmoquine.

Dr. Kikuth said that he was " unable to commend Simeons' practice of injecting the total daily quantity of atebrin musonat at one dose. Intravenous injections, both of atebrin musonat and quinine were superfluous and dangerous." *W. Fletcher.*

GILL (C. A.). **Some Points in the Epidemiology of Malaria arising out of the Study of the Malaria Epidemic in Ceylon in 1934–35.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. Feb. 29. Vol. 29. No. 5. pp. 427–466. With 17 figs. [23 refs.] Discussion pp. 466–480. With 2 maps & 3 charts.

The author's theory is that the epidemic began with a fulminating outbreak of relapses among healthy carriers due, apparently, to an excessive increase in atmospheric humidity. The abnormal number of anopheles was not the cause, though it was partly responsible for the magnitude of the epidemic.

Colonel Gill's official Report on the Ceylon epidemic has been summarized already in this *Bulletin*, 1936, Vol. 33, p. 214 ; in his present paper he deals more particularly with its causes. He emphasizes " the powerlessness of modern medical science to prevent the outbreak of malaria epidemics or to check materially their course," though in his Report he implied that future epidemics in Ceylon might be warded off by antimalarial measures undertaken during the inter-epidemic period. He is of opinion that little can be done to control malaria except in towns and closed communities ; " what of the large free population of rural areas, numbering more than 80 per cent. of the total ? . . . despite the great advance of knowledge during the past 50 years, the control of malaria, as a practicable proposition, is almost as far to seek as it was a century ago." In the discussion which followed the paper, this pessimistic view was criticized by Sir Malcolm WATSON who instanced the success of antimalarial work carried out by himself in a rural area of the Malay States.

Colonel Gill states that the great malaria epidemics of the Punjab, Mauritius and other places have been associated with particular sun-spot phases. " In Ceylon, the association of malaria epidemics with the epoch of maximum and minimum sun-spots is extremely close. . . . For the present, it will suffice to state that the relationship of malaria epidemics to the sun-spot cycle is sufficiently close to permit of the conclusion that the cyclical periodicity of malaria epidemics is attributable to the changes in world-weather associated with periodic oscillations in solar activity."

Special weather conditions are associated with malaria epidemics ; in the pre-epidemic period there is a rise of atmospheric humidity. In the wet zone of Ceylon there was a failure of the south-west monsoon with a progressive decline of atmospheric humidity during July, August and September, with the result that the rainfall in October, though it was below normal, occasioned a sharp rise in humidity, which occurred at the end of September, three weeks before the outbreak of the epidemic. The severity of the epidemic was greatest where this rise was greatest. It was very high, for example, in Kurunegala, the focal centre of the epidemic.

Such a change in humidity appears to cause " a change in the relationship of the malaria parasite and the human host, whereby, at the commencement of an epidemic, an ' epidemic of relapses ' is precipitated amongst human carriers." Why does Colonel Gill believe

that the Ceylon epidemic began as an epidemic of relapses? In the first place, it began with fulminating suddenness; "not only was the onset of the epidemic remarkably abrupt but . . . it could often be fixed to a day. Thus, at the Civil Hospital, Kurunegala, the daily average number of attendances, which had fluctuated between 130 and 150 per diem for several months before the epidemic, was more than doubled on 29th October, 1934, with the result that the total attendances, which in the week ending 27th October were 1,305, numbered 2,763 at the end of the following week . . . it was decided to confine attention to a single locality and Kurunegala town, population 10,500, was selected for this purpose, partly because it was the most severely affected large town in the island, and partly because exceptionally complete statistical data were obtainable." A second point in favour of the relapse theory was that very few children suffered during the first few weeks of the outbreak. ". . . it is difficult to believe that the morbidity amongst adults during this period could have been mainly caused by new infections, since it would imply that infected anophelines had selected adults alone for attack. Furthermore, . . . it would be necessary to assume that a large number of anophelines had become infected from such human carriers as existed during a period of good health, and had thereafter dispersed, and, in due course, infected a large number of persons (*excluding children*) about the same time, who, after an identical incubation period, commenced to fall ill together. This sequence of events is too improbable to call for serious consideration, and it is therefore held that the sickness during the first month of the epidemic was mainly due to an 'epidemic of relapses' amongst apparently healthy human carriers."

The next step in the epidemic was the spreading of the infection from the relapsed cases to the surrounding non-immune population by an abnormal abundance of anopheles. *A. culicifacies*, in normal times, is not prevalent in the Wet Zone, where the epidemic occurred, it is much less common there than in the Dry Zone, but, in 1934, it was extremely prevalent much to the south of its normal habitat and bred freely among the pools in the beds of the dwindling rivers. The coincidence of the following was responsible for the magnitude and severity of the epidemic: (1) A large number of relapses following an abnormal rise in atmospheric humidity associated with cyclical sun-spot changes; (2) An abnormal prevalence of the carrier species of anopheline, *A. culicifacies*; (3) A non-immune population suffering from privation due to the drought.

In the discussion which followed, Colonel S. P. James brought to notice a suggestion made to him by Dr. de Verteuil of Trinidad that the onset of rain after the 1934 drought in Ceylon might have caused a migration of anopheles from the malarious Dry Zone into the Wet Zone and that this was responsible for the epidemic. Migration on a small scale occurs annually in Trinidad, but, at intervals, and after a prolonged drought, migration occurs on an immense scale.

W. F.

Ellison (F. O'B.). **Malaria Epidemics and Sun-Spot Cycles.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. Apr. 8. Vol. 29. No. 6. pp. 659–665.

"Colonel Gill (above) has suggested that a definite correlation exists between the maximum and minimum development of

sun-spots and epidemics of malaria. . . . The relation between malaria epidemics and sun-spot activity is not as close as Gill would have us believe either in Ceylon or elsewhere. The last two outbreaks in Ceylon, 1934 and 1928, do, it is true, coincide with a minimum and a maximum of sun-spots, but from 1923 back to 1905, epidemics both in Ceylon and elsewhere, taking Gill's own dates with one or two necessary corrections, have prevailed during one and a half complete sun-spot cycles with absolute impartiality at sun-spot maximum, minimum and every stage in between." *W. F.*

JAMES (S. P.), NICOL (W. D.) & SHUTE (P. G.). **Clinical and Parasitological Observations on Induced Malaria. (With Notes on their Application to the Study of Malaria Epidemics by** S. P. JAMES).—*Proc. Roy. Soc. Med.* 1936. June. Vol. 29. No. 8. pp. 879–893 (Sect. Trop. Dis. & Parasit. pp. 27–41). With 16 figs.

[This is a very important and interesting paper. Colonel James's theory of the Ceylon epidemic, based upon experiments with the Madagascar strain at Horton Mental Hospital, seems to be as follows: There was a great increase in the number of anopheles; the population was non-immune. Malaria increased gradually until a point was reached where transmission among the highly susceptible population was being repeated at a great rate. This procedure (as shown by observations at Horton) results in a fundamental change in the nature of the endemic strain of parasite which becomes converted into an epidemic strain; the special attribute of such an epidemic strain is its reproductive vigour—it increases much more rapidly in the blood, and it produces many more gametocytes. This produces a far heavier infection of anopheles, and, consequently, a much heavier dose of sporozoites of the vigorous epidemic strain is injected into the host. This is responsible for the severe type of disease characteristic of epidemic malaria.

Colonel James made the interesting statement during his lecture, that atebrin acts as a causal prophylactic, destroying the sporozoites of benign tertian malaria.]

Epidemic malaria and endemic malaria differ from one another clinically; endemic malaria is comparatively mild and seldom kills, but epidemic malaria is severe and often fatal. What is the reason? It is generally attributed to lack of resistance due to poverty, famine and the like which sometimes, as in Ceylon, happen to coincide with the epidemic. The author's work at Horton has led him to other conclusions; he ascribes the clinical malignancy of epidemic malaria rather to a change in the nature of the attacking parasite than to a change in the defensive powers of the host. "The change in the parasites which we have observed at Horton (and which we think may be the change that is responsible for the severity of epidemic malaria) is not an increase of virulence, but an increase of physical vigour and vitality leading to the production of individuals which represent the species in its perfect form. In these individuals the normal processes of reproduction, sexual as well as asexual, are carried on in the most regular and complete manner of which the species is capable. . . . the number of parasites . . . exceeds considerably the number which appears in the blood of persons infected with a less active strain . . . and the result of using a strain which reproduces itself freely and vigorously is that non-immunes of normal

susceptibility who are infected with it develop the severe epidemic type of the disease." Colonel James did not describe in detail the technique by means of which he brought the endemic "Madagascar strain" to the degree of physical vigour and activity in which it caused cases of the severe epidemic type, but an essential part of this technique consisted in passing the strain repeatedly through persons who possessed no resistance to malarial infection. When this change to epidemic type has been effected, the strain produces gametocytes earlier and in greater numbers. Normally, no gametocytes are found during the first five days in patients infected with the Madagascar strain, and in only 33 per cent. are they found during the first week; but, after the strain has been passaged according to Colonel James's "plan," gametocytes are found in some cases as early as the third day, and in 82 instead of 33 per cent. they are found during the first week. The number of gametocytes in the blood is much more numerous after the "plan" than before it and mosquitoes are much more easily and heavily infected; it was with this object that the "plan" was originally devised. As regards the clinical change, after the "plan," the onset of fever is more like that seen in a subtertian infection than in benign tertian; it begins as an irregular fever, without rigors, which increases daily until, by the 5th or 6th day, there are one or two paroxysms every day; finally parasites become so numerous, and the patient becomes so ill, that the attack must be cut short by quinine. There is usually a relapse or "recrudescence" 3 weeks later, and one or two more afterwards at similar intervals. Subsequently, there is a long period of freedom, and then, suddenly, a sharp recurrence at about the 7th month which may be followed by several recrudescences. How can a relapse be distinguished from a primary attack? By the temperature chart, and by blood films. A relapse begins abruptly with a rigor, instead of gradually like a primary attack; subsequently, there are paroxysms every other day, instead of every day, and gametocytes are present as early as the first or second day.

Colonel James next considered the dose of infection. Infection by the bites of two or three lightly infected mosquitoes is usually quite different from the result of infection by the bites of many heavily infected insects. When only a small dose of sporozoites is injected the result is an abortive primary fever which passes off in two or three days, or there is no primary attack at all, and the infection remains latent. In either case, seven months later, at a period corresponding with the period of recurrence in the severe type, there is a sudden sharp attack. This observation is a repetition in the laboratory of what happens in countries like Holland where (because only one or two lightly infected mosquitoes are responsible for each indigenous case) most of the cases are abortive or latent in the primary attack and are only detected at the period of recurrence in the following spring. In benign tertian malaria, the condition of latency can always be produced by a few prophylactic doses of *atebrin which kills most, but not all, sporozoites* injected by a mosquito. The recurrences, or late relapses, which appear 7 months after the primary attack of benign tertian, occur only in cases infected by the bites of mosquitoes, they are never seen in cases induced by the inoculation of malarial blood. Recurrences are not in any way dependent on climate, season or atmospheric humidity. "In India, the onset of recurrences between six months and a year after anti-relapse schemes of treatment upsets

cherished opinions based on a period of observation of only two months." (See SINTON below.)

The annual summer-autumn epidemic of malaria in Europe begins with benign tertian and becomes predominantly subtertian later. What is the explanation of this? Malignant tertian malaria is an acute disease comprising a primary attack followed by several recrudescences at short intervals. There is no recurrence, the total duration of febrile manifestations seldom exceeds 6 months and is usually less. By the time anopheles have become prevalent in the early summer, nearly all the cases of malignant tertian have ceased to relapse, but cases of benign tertian are still having recurrences and recrudescences; gametocytes are plentiful in the blood of these cases, anopheles readily become infected and consequently benign tertian predominates.

Colonel James next viewed the Ceylon epidemic in the light of the observations already recounted. First, he dealt with the question whether the epidemic was initiated by an outburst of relapses or whether it started with an increase of primary cases, and, as evidence, he took the temperature charts and the blood films. Sixty-three charts from the Kandy Hospital, recorded in the early part of the epidemic before the middle of November, showed only 6 which represented recrudescences or relapses; the remainder were charts of primary cases with one or more febrile paroxysms every day. As regards the parasitological evidence on this point, Colonel James considered a study of blood films made by Dr. WIJERAMA at Colombo. This observer found no gametocytes during the first 6 weeks of the epidemic and this "is strong evidence for the view that most cases during that period were primary attacks." As confirming this view Colonel James considers the evidence of Dr. P. B. FERNANDO of Colombo who noted that a feature of the epidemic was the uncommonly heavy parasitic infections met with. "This accords with what I said in the earlier part of my paper about the cause of the severity of primary attacks in cases infected with our Madagascar strain."

The next question was whether the epidemic began suddenly or gradually. Here again, Colonel James decided on the evidence of Drs. WIJERAMA and FERNANDO. "The curve starts with cases admitted during the week ending October 7th when one film positive for *P. vivax* and one for *P. falciparum* were recorded." After a period of gradually rising prevalence lasting about 8 weeks, both types began to rise sharply and, a week or two later, malignant tertian malaria evidently became severely epidemic. "It seems as if we must decide that the epidemic began gradually rather than suddenly and that it did not become explosive in character until it had been increasing steadily for six weeks or more." The low mortality at the beginning of the epidemic, next the rise of mortality in children, and later in adults, are considered to be consistent with the view that the endemic strain gradually attained its maximum vigour in the same way as it was attained experimentally by the Madagascar strain. The course of the Ceylon epidemic was not different from epidemics in other places where the bulk of the population possessed little immunity. For example, the town of Nemi, near Rome, is ordinarily free from malaria and anopheles are very rare; but, when Lake Nemi was lowered 14 metres in a search for Caligula's barges, numerous breeding places were created, anopheles became very abundant and there was an epidemic of malaria. "This epidemic, like the epidemic in Ceylon, showed that in an area where there are few gametocyte carriers and

almost no anopheles, an invasion by great numbers of these insects can give rise to a serious epidemic provided the population at risk possesses no immunity to the disease. It is equally true that areas in which there are plenty of anopheles, but no gametocyte carriers, may almost immediately suffer from an epidemic when a number of gametocyte-carrying cases are imported." *W. F.*

GILL (C. A.). **The Mode of Onset of the Malaria Epidemic in Ceylon.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. June 30. Vol. 30. No. 1. pp. 101–107.

A study of temperature charts and of records of blood examinations received from Ceylon led Lt.-Col. S. P. JAMES to the conclusion that the 1934–35 epidemic of malaria in that island started gradually and was associated with a slow, steady increase of primary infections. Colonel Gill, on the other hand, is of the opinion that the epidemic started suddenly by means of an "epidemic of relapses" [*ante*, pp. 213–220, and also above].

In the paper under consideration Colonel Gill reproduces tables of hospital and dispensary returns, and of blood examinations of residents of Colombo, which, he considers, supports his thesis that the onset of the epidemic was associated with a sudden outbreak of relapses which was followed about one month later by a sudden outburst of primary infections. *Norman White.*

FERNANDO (P. B.) & SANDARASAGARA (A. P.). **A Clinical Study of 647 Patients treated for Malaria during the Ceylon Epidemic of 1934–1935.**—*Ceylon Jl. Sci.* (Sect. D. Med. Sci.) 1935. Dec. 3. Vol. 3. Pt. 4. pp. 195–233. With 1 fig. [13 refs.]

Relapses are more frequent after atebrin than after quinine. Atebrin is more toxic than quinine or plasmoquine.

The 647 cases were under the authors' care in the General Hospital, Colombo. Benign tertian was common at the beginning, but during the explosive stage of the epidemic subtertian infections predominated; as it declined the proportion of benign tertian cases increased. The case mortality was 4·3 per cent. but more than a quarter of the fatal cases were moribund on admission. There were 54 cerebral cases, and 19 of an oedematous type which appeared to belong to the same category as war oedema or starvation oedema. The patients were treated as follows (according to Table I which does not include all): (1) Quinine 21 grains daily for 7 days, or for period of stay in hospital,—220 cases. (2) Atebrin 0·3 gram daily for 5 days—144 cases. (3) Atebrin and quinine in the above doses for 5 days—39 cases. (4) The same, with 0·02 gram of plasmoquine daily for 5 days—28 cases. (5) Quinine 21 grains daily and plasmoquine 3 grains daily—10 cases. [This is probably a misprint, 3 grains is equivalent to nearly 0·2 gram.]

The authors concluded that quinine was superior to atebrin in bringing down the temperature and in preventing relapses. There were about 3 times as many relapses after atebrin as after quinine. Toxic symptoms occurred in 3·3 per cent. of 299 cases treated with atebrin and "one patient died as the result of atebrin poisoning." In another, pain starting on the 5th day was severe enough to require morphia; two patients collapsed after 3 days' treatment [the details

of the fatal case are not very convincing ; the patient had received, apparently, only 6 tablets]. The authors consider atebrin contra-indicated in cases of kidney disease, and intravenous quinine contra-indicated when the blood pressure is below S.90. They conclude that quinine is still the most valuable anti-malarial drug available and that atebrin is more toxic than quinine or plasmoquine. Plasmoquine they found non-toxic when given in doses of 0·02 gram daily for 5 to 8 days. *W. F.*

SINTON (J. A.). **A Discussion of Some Points contained in the " Third General Report of the Malaria Commission " on " the Therapeutics of Malaria."**—*Quarterly Bull. Health Organisation, League of Nations.* Genevà. 1935. Dec. Vol. 4. No. 4. pp. 643–707. [25 refs.]

The author attacks the Commission for saying that it is useless to give plasmoquine or alkalis with quinine. He regards much of the advice given in the Report as impracticable or dangerous to life.

His first criticism is that while it is stated on the first page of the Report [this *Bulletin*, 1933, Vol. 30, p. 837] that it is based on the findings of research workers in several countries as the result of their experience with cinchona alkaloids and synthetic remedies in the field, yet, as one reads further, one finds that the bulk of the work to which prominence is given was conducted under laboratory conditions in mental hospitals, and further, almost at the end of the Report, one finds the statement that the subject is dealt with " from the point of view of persons who are in a position to obtain expert medical advice and efficient care *rather than from that of the mass of the population of malarious countries.*" While he admits that the Report is most valuable to a certain class of research worker, it has come to his notice that, in many instances, it has hindered rather than helped the clinician in the tropics.

Colonel Sinton has reported excellent results in the prevention of relapses by the administration of a combination of quinine and plasmoquine. The Commission in their Report consider that this treatment is of doubtful value, and that the claim that giving plasmoquine in addition to quinine prevents relapses " can only be described as paradoxical." In confutation the author cites the results of this treatment at Kasauli :—

" In the early days of the Malaria Treatment Centre, when intractable chronic relapsing cases of malaria were sent to Kasauli from all over India, these were mainly benign tertian infections. In those days when the value of different cinchona alkaloids was being tested, the Centre had usually about 200 patients and a waiting-list of many others requiring admission. Since the introduction of the combined quinine and plasmoquine treatment as a routine in the army, more especially for the treatment of such quinine-intractable cases, the numbers fell so low that the Centre had to be closed. . . . by this means a much higher percentage of permanent cures can be obtained under certain conditions than with quinine therapy or with plasmoquine alone in non-toxic doses. . . . In the work of the Malaria Treatment Centre it was shown that the combined treatment with plasmoquine reduced the number of parasitic and clinical relapses in chronic benign tertian infections to about 10 per cent., during an observation period by blood examination of at least 8 weeks after the termination of treatment. On the other hand, with any course of treatment with the cinchona alkaloids alone, the relapse rate in this type of case averaged about 65 per cent. in the same period."

The daily dose of plasmoquine recommended is 0.015 gram under medical supervision.

The Commission states in its Report that, owing to lack of knowledge of the natural course of benign tertian malaria and of the most important type of relapse which occurs about 8 months after the primary attack, "the results of all clinical tests conducted prior to 1931 were quite misleading." The author writes: "From a consideration of the objections raised by the Commission in its Report, it does not appear to me that such a sweeping statement is justified in the case of the infections studied at the Malaria Treatment Centre, Kasauli, before 1931. There is considerable evidence to show: . . . (2) That the patients were observed under much more natural conditions than in mental hospitals; (3) That, with the method of observation used, the occurrence of 'late' relapses in the 'natural course' of benign tertian malaria does not invalidate the results obtained to any marked degree; and (4) That the findings recorded have proved of much practical value to workers in India." He suggests that, even if one admits that late relapses are a normal event and not a peculiarity of the particular strains with which the Commission worked, the infection would have been detected during the regular routine thick-film examinations made during the first 8 weeks after the cessation of treatment at Kasauli. With reference to subtertian malaria, he writes:—

"In condemning or depreciating the results recorded with the 'quinine and alkali' treatment of malignant tertian malaria, the Commission appears to have overlooked its own statements—that different drugs may react differently on different strains, and to have allowed its judgment to be biased by the results of experiments with a few very virulent strains of *P. falciparum*. In view of the great differences in the reaction of different strains to therapeutic agents, I cannot understand why the Commission is unable to accept the results of our Indian work."

It is suggested in the Report that relapses are more easy to cure than primary attacks, but most of the author's Kasauli patients had had 3 or 4 febrile relapses before they came to him, yet 65 per cent. relapsed after treatment with quinine—*i.e.* appreciably higher than the 50 per cent. mentioned by the Commission as occurring in primary infections receiving much less treatment.

The Commission deprecates the employment of a standard treatment and advocates that each individual case should be treated on its merits, taking into consideration the virulence of the strain of parasite, the degree of immunity and the stage of disease, whether primary attack or relapse; but, says the author:—

"Unfortunately, I am unaware of any methods by which this can be done either clinically or microscopically under natural conditions . . . there are very few places in the tropics or other malarious countries where it would be possible to carry out . . . the lines of investigation recommended by the Commission, because one would have to assume that *all the strains* of one species of *Plasmodium* were identical in a given area, for there is no known method of differentiating these strains by their morphological characters. Such an assumption does not seem justifiable. . . . The patient may be infected with multiple strains of the same species of parasite . . . it is at present, impossible under natural conditions, or in the laboratory, to determine with any scientific accuracy whether the attack being studied is a relapse, a reinfection with a homologous strain, or a superinfection with a heterologous one."

In Sinton's "standard treatment" quinine is given in those doses which are considered necessary to cure radically within a week the majority of the strains of parasite. If the patient develops a relapse he is given a treatment which contains plasmoquine in combination with quinine. Some "standard treatment," urges Colonel Sinton, is an essential measure, if one is to obtain the greatest benefit of the greatest number of patients for the money available.

The Commission states that quinine has but little effect when given on the first or even the second day of the initial fever. They also state that in hospitals where there is insufficient watchfulness the fatality due to their strain of benign tertian is not less than 10 to 14 per cent. Colonel Sinton draws attention to the danger of undetected mixed infections, and asks :—

"If delayed treatment in benign tertian infections can cause such an excessive mortality, even among patients receiving ordinary care in hospital, how much more dangerous is such a plan of treatment likely to be in general practice ? . . . If treatment be withheld on the assumption that the primary diagnosis of *P. vivax* is correct, the rapid development of *P. falciparum* may remain undetected until therapeutic measures are too late to save the life of the patient. . . . However desirable such 'delayed treatments' may be for the purposes of researches into the value of certain therapeutic agents, . . . I do not consider that it is a measure which can justifiably be recommended for use in the majority of hospitals in the tropics, nor for the practising physician of any country, in our present state of knowledge."

If the development of immunity is favoured by delaying treatment, it is an immunity against the infecting strain only, and will give little or no protection against other strains.

The Commission recommend different treatments for primary attacks and for relapses. They suggest that as there is little danger to the patient's life in a relapse he should be allowed to have several paroxysms before treatment is begun, and that the amount of this treatment should be small, in order that the natural process of premunition may not be interfered with. Colonel Sinton points out that a patient who returns with a malarial attack some time after the termination of treatment for the primary attack is not necessarily suffering from a relapse, it may be a fresh infection or superinfection with a heterologous strain or species of parasite. "If such a case be treated by the method which is recommended by the Commission as suitable for a recrudescence or a relapse . . . it may be too late to save the patient's life when proper treatment is instituted."

W. F.

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

SHRIMPTON (E. A. G.). **A Survey of the Incidence of Relapsing Fever in China.**—*Chinese Med. Jl.* 1936. Feb. Supp. No. 1. pp. 312–344. With 6 figs. (1 map). [25 refs.]

A general account of the epidemiology of relapsing fever in China based on data obtained in the "Survey of Hospital Patients" for 1933 and 1934, and also from hospital reports and references to the literature.

The disease has been reported from most of the provinces of China, but there are no well defined endemic areas. In typical areas a marked seasonal distribution is found with a maximum in May and June, but under abnormal conditions, such as famines, civil disturbances, overcrowding, etc., this maximum may occur in other months. The distribution shows an intimate connexion with temperature, probably correlated with changes in the clothing habits of the population in relation to climate. The disease is almost entirely confined to the lower grades of society where overcrowding and unhygienic surroundings are manifest.

There is a ratio of 4 males to 1 female, which is related to the degree of exposure to contagion resulting from the economic situation in the larger towns. The age distribution after 15 years shows a slightly higher expectation, probably due to the effects of different exposures to infection. No cases were recorded in infants under a year old, and from 1 to 15 they were relatively infrequent.

The lower incidence of the disease in the south may be ascribed to the smaller number of reservoirs of infection, and to the shorter period of favourable conditions for the spread of the vector.

A marked alteration in the number of reported cases each year is observed and it is suggested that this may be due to the immunity from infection lasting for only a short period. *E. Hindle.*

COLES (Alfred C.). **Observations on the Life-History of the Spirochaetes of Relapsing Fever.**—*Jl. Trop. Med. & Hyg.* 1936. Apr. 1. Vol. 39. No. 7. pp. 77–81. With 12 photomicrographs & 7 figs.

The author examined dried films of *Spirochaeta pallida*, *S. duttoni*, *S. vespertilionis*, *S. anserina* and also *S. buccalis* and other mouth spirochaetes, stained in various ways, and in all cases found evidence of the existence of small granules within the spirochaetes.

The air-dried films were fixed in alcohol and then stained in well-diluted Giemsa for 12, 24 and 48 hours respectively. After washing and drying, half the stained films were differentiated by dropping on a few drops of Orange-tannin and after allowing this to act for 15 minutes the preparations were washed in water. The films were examined both with ordinary and dark-ground illumination. In addition Burri's Indian ink method was used and found to show the existence of beads or dots in all the spirochaetes examined.

The author concludes from his observations that spirochaetes consist of a tube containing minute granules and that under certain conditions the wall of the organism bursts and the granules are discharged.

These infective granules would be so small that they would be almost indistinguishable from other minute granules present in the blood and tissues.

[BALFOUR and the reviewer* both described this process in the case of *S. anserina*, nearly 25 years ago, and the latter advanced a similar explanation of the so-called invisible infecting stage of spirochaetal infection (see this *Bulletin*, 1912, Vol. 1, p. 34).] *E. H.*

VINZENT (R.), SEGUIN (P.) & DAUFRESNE (M.). Technique d'isolement des spirochètes commensaux de l'homme. [**A Technique for the Isolation of the Commensal Human Spirochaetes.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 5. pp. 406–408.

A modification of Noguchi's technique by means of which the authors have been able to isolate in pure culture various commensal spirochaetes from the human mouth, lungs, intestine and genitalia. The medium consists of one part of sheep serum mixed with three parts of nutrient agar, the mixture then being placed in tubes 11 mm. in diameter and 22 cm. long, each containing a piece of fresh sterile kidney from a guineapig, rabbit, or sheep. The nutrient agar is prepared as follows :—

Meat extract (500 gm. in a litre of water) ...	1,000 cc.
NaCl	5 gm.
Agar	15 gm.

adjusted to pH 8.

This medium can be used immediately and also preserves its properties for a month.

The tubes are inoculated by means of a capillary pipette passed down the centre of the medium to the neighbourhood of the piece of kidney, care being taken to avoid the formation of bubbles. The medium is then covered with a layer of sterile vaseline and kept at 37°C. About the 5th day or sometimes earlier, subcultures are made. The selected tube is cut across about 2 or 3 cm. above the piece of tissue and then by means of a pipette a small cylinder of the medium is isolated from the region between the path of the inoculation and the wall of the tube. This material can be examined by means of dark-ground illumination and if it contains spirochaetes is inoculated into a fresh tube in the neighbourhood of the kidney. This process is repeated indefinitely until the spirochaetes are separated from contaminating bacteria. Sometimes the process can be accelerated by passages in semi-coagulated serum containing kidney, to which is added 2 to 4 drops of a 1 per cent. solution of malachite green in each tube. The dye retards the multiplication of the bacteria without affecting the spirochaetes.

The separation of spirochaetes belonging to different species is more difficult and both biological and mechanical methods have to be employed. Some spirochaetes grow faster than others, and also the medium can be modified in various ways, such as by altering the pH, omitting the sterile tissue, using ascitic fluid instead of broth, etc. The mechanical methods follow the technique indicated by VEILLON for the isolation of anaerobic bacteria. *E. H.*

* HINDLE, E. (1912). *Parasitology.* Vol. 4. p. 463.

SEGUIN (P.) & VINZENT (R.). Étude systématique des spirochètes buccaux d'après les caractères de culture. [**A Systematic Study of Buccal Spirochaetes based on their Cultural Characteristics.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 5. pp. 408–411.

The authors give the characteristics of the following species of buccal spirochaetes isolated in pure culture from the human mouth by the technique described above :—

S. microdentium Noguchi (1912) ; *S. ambigua* n. sp. ; *S. commandoni* n. sp. ; *S. skoliodonta* Hoffmann 1920 ; *S. trimerodonta* Hoffmann 1920 and *S. macrodentium* Noguchi 1912 (= *S. tenuis* Gerber). They were unable to obtain a pure culture of *S. buccalis*. *E. H.*

LI YUAN PO. Méthode de préparation simplifiée de mon milieu pour la culture du *Spirochaeta recurrentis*. [**A Simplified Method of preparing my Medium for the Culture of** *S. recurrentis*.]—*Ann. Parasit. Humaine et Comparée.* 1936. Jan. 1. Vol. 14. No. 1. pp. 76–77.

A modification of the original method of preparing this medium [see this *Bulletin*, 1933, Vol. 30, p. 366]. The yolk of an egg, of which the shell is previously sterilized with alcohol, is mixed thoroughly with 400 cc. of sterile 0·85 per cent. saline solution in a conical flask. The egg must be fresh and preferably with the shell of a dark brown colour. About 5 cc. of this mixture is added to each tube containing egg-white coagulated at 80°–85°C. for twenty minutes. The tubes are kept at 56°C. for two hours and then a layer of sterile paraffin is added. After cooling the tubes are ready for use in spite of their opacity. The medium has been found to give excellent results, especially with *S. recurrentis*, and one can count on 100 per cent. success in cultures from human blood if the operation is conducted aseptically. For subcultures it is necessary to add one or two drops of citrated blood to each tube.

E. H.

ADLER (S.), THEODOR (O.) & SCHIEBER (H.). **Transmission of Relapsing Fever by Ticks in Palestine.** [Correspondence.]—*Lancet.* 1936. Feb. 22. p. 448. Also in *Harefuah.* Jerusalem. 1936. Mar. Vol. 10. No. 3 (57). [In Hebrew. English summary p. 1.]

A recent investigation of a small outbreak of relapsing fever in Kfar Vitkin, south of Hedera in the coastal plain, showed that all infections could be traced to a cave infested with *Ornithodorus papillipes*. Ticks collected in the cave readily transmitted spirochaetes to rats in the laboratory. This tick produces a local analgesia while biting and coxal fluid and faeces are not excreted during feeding. The transmission, therefore, is obviously by the bite, and the authors were able to demonstrate the presence of spirochaetes in smears made from the puncture wounds both of a human subject and a rat bitten by infected ticks.

E. H.

KOHLS (Glen M.) & COOLEY (R. A.). **Notes on the Occurrence and Host Relationships of the Tick** *Ornithodorus talaje* **in Arizona.**—*Public Health Rep.* 1936. Apr. 24. Vol. 51. No. 17. pp. 512–513.

Larval specimens of *Ornithodorus talaje* were collected on kangaroo rats (*Dipodomys* sp.) in an uninhabited region 40 miles southwest of

Aguila, Arizona, and nymphs and adults from soil removed from their burrows. Pocket-mice and jack rabbits in the same localities carried no ticks and therefore the authors suggest that in this region *O. talaje* may be distinctly limited in its host relationships. *E. H.*

LIPSTEIN (I.). Transmission de *Spirochaeta novyi* par *Pediculus corporis*. Contribution à la technique de l'élevage des poux. [**The Transmission of** *Spirochaeta novyi* **by** *Pediculus corporis*. **Contribution to the Technique of rearing Lice.**]—*Ann. Parasit. Humaine et Comparée.* 1936. Mar. 1. Vol. 14. No. 2. pp. 113–125. With 5 figs. on 1 plate. [23 refs.]

The author employs the usual leather " bracelet " method of feeding the lice contained in pill-boxes, but states that they live better if kept between meals in a glass jar over damp cotton wool in an incubator, at 35°C. during the night, and either at 24°C. or 35°C. during the day.

The lice were infected by feeding on the shaved ventral surface of a rat infected with *S. novyi*, but about 90 per cent. of the lice died after such a meal, animal blood being generally toxic to them.

The spirochaetes in the gut of the lice were found to become immobile after about 3½ hours and soon disappeared. After 24 hours no trace of them could be found in the body of the lice.

Infected lice were ground up and inoculated into rats at varying intervals. They were found to be virulent 24 hours and 7, 8 and 9 days after the infective meal, but after 48 hours and 5 days were not virulent. It is curious that when the spirochaetes were inoculated directly from one rat to another the infection was always fatal, but the infections produced by the emulsions of lice were generally benign. *E. H.*

SERGENT (André). Passage dans le lait du spirochète de la fièvre récurrente hispano-africaine (souche algérienne). [**The Passage in the Milk of the Spirochaete of Spanish-African Relapsing Fever (Algerian Source).**]—*C. R. Soc. Biol.* 1936. Vol. 122. No. 17. pp. 213–214.

Four guineapigs were infected with an Algerian strain of *S. hispanica* and at the height of the infection their milk was collected and inoculated subcutaneously into a series of guineapigs, all of which developed the disease.

Although the infection was thus shown to be present in the milk of all four animals, spirochaetes were never found in the milk by microscopical examination. Out of the seven young guineapigs which fed on this infected milk only one developed the disease, after an incubation period of 14 days. *E. H.*

KRÖBER (Friedrich). Einige seltene Beobachtungen bei afrikanischem Rückfallfieber. [**A Few Notes on African Relapsing Fever.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1936. Apr. Vol. 40. No. 4. pp. 160–165.

The author gives two examples of intra-uterine infection with relapsing fever. In the first case the mother developed the disease the day after her child was born. The infant, although healthy at birth and its blood negative, 5 days later was found to be heavily infected with spirochaetes and in spite of treatment succumbed to the disease. In

the second case the infant was born of an infected mother and its blood remained negative, but on the third day it became unwell and as a precautionary measure was treated with myosalvarsan. The following day the child was better and remained well and the author reasonably assumes that it had probably been infected at birth and the treatment arrested the development of the disease.

With regard to cases in which patients show recurrence of attacks within a few weeks of receiving a course of treatment, the author adduces evidence to show that these must be regarded as new infections, rather than examples of the development of salvarsan-resistant strains of spirochaetes.

Finally, the immunity problem is briefly discussed, and it is considered that treatment prevents the full development of any immunity against the infection; but, nevertheless, it is generally inadvisable to delay treatment as occasionally the disease may be rapidly fatal.

E. H.

CALWELL (H. G.). **A Case of Congenital Relapsing Fever.**—*East African Med. Jl.* 1936. Feb. Vol. 12. No. 11. pp. 347–348.

The record of a case of infection with *S. duttoni* in a child, who developed the disease on the ninth day after birth and died on the 12th day. The mother showed symptoms of the disease on the third day of the puerperium, and since the infant was not at any time exposed to the bites of infected ticks, the infection seems to have reached the child either through the placenta or in some other way during parturition.

E. H.

SERGENT (André). Caractères pathogènes d'une souche algérienne du spirochète de la fièvre récurrente hispano-africaine. [**The Pathogenic Characters of an Algerian Strain of Spanish-African Relapsing Fever.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 14. pp. 1520–1522.

An experimental study of an Algerian strain of *Spirochaeta hispanica*; various animals were inoculated subcutaneously, in order to determine their susceptibility to this infection.

The pig, donkey and cat were found to be refractory but the other animals tested were susceptible in varying degrees. The guineapig is regularly infected and always shows an acute febrile attack followed by a chronic state of infection. The rabbit, 17 out of 18 infected, shows a similar type of infection to the guineapig, but the parasites are present in the blood for a shorter time and in fewer numbers. The white mouse becomes infected after 24 hours' incubation, but the attack is of short duration and the animals recover. Monkeys showed an attack of 4 to 5 days and in 1 out of 3 cases there was a relapse 10 days later which lasted for 5 days. Five out of 6 dogs were infected; two young ones showed spirochaetes in the blood for 2 and 7 days respectively; the adults showed latent infections. The fowl is also susceptible but shows no obvious spirochaetes in its blood. Nevertheless its blood becomes infective to guineapigs after about 5 days' incubation and this infectivity persists for about 3 weeks.

E. H.

SERGENT (André). Épreuve de la prémunition croisée appliquée à quelques souches algériennes de spirochétose hispano-africaine. [**Cross-Immunity Tests applied to Various Algerian Strains of Spanish-African Relapsing Fever.**]—*Bull. Soc. Path. Exot.* 1936. Mar. 11. Vol. 29. No. 3. pp. 245–251. With 2 figs.

The author made cross-immunity tests with 3 Algerian strains of *Spirochaeta hispanica* in guineapigs. One strain had been isolated from a human case of infection at Chiffalo, a second from *Rhipicephalus sanguineus* in the same locality, and both strains had been kept in the laboratory for two and a-half years. These two were found to be serologically identical. A third strain, isolated from a human case in another district only 3 months previously, belonged to the same species, but was more virulent. *E. H.*

SERGENT (André). Emploi thérapeutique du sérum de convalescents de fièvre récurrente hispano-africaine. (Étude expérimentale.) [**The Therapeutic Use of Convalescent Serum in Spanish-African Relapsing Fever. An Experimental Study.**]—*Bull. Acad. Méd.* 1936. Mar. 17. 100th Year. 3rd Ser. Vol. 115. No. 11. pp. 463–467.

The author has studied the value of convalescent serum both for protection and treatment using guineapigs infected with *Spirochaeta hispanica.* This is an ideal animal to use, for out of 1,100 guineapigs inoculated with this strain, not one has been refractory to the infection.

In one series of experiments 5 cc. of convalescent serum was injected into guineapigs either the day before, the same day, or the day following the inoculation of spirochaetes. In all three groups the serum prolonged the incubation period and attenuated the attack, but on the other hand the infection was prolonged with many small relapses.

With regard to the curative action, out of 71 infected guineapigs 25 were left untreated and the remaining 46 inoculated with convalescent serum either the first, second, third or fourth day of the attack. In all cases the serum caused a sudden fall in temperature and the number of parasites within 24 to 36 hours. The effect was most marked on the third day of infection when the average duration of spirochaetes in the blood was reduced to an average of only 1·5 days, as compared with 8 days in the controls.

Convalescent serum collected from guineapigs after the end of the first attack was found to be more effective than after several weeks. The action was found to be proportional to the dose injected and better results were obtained by giving a single large dose, than a number of small doses.

From these results the author is of the opinion that human cases of this disease should be treated by means of convalescent serum, for this infection does not respond to arsenical treatment and the disease is often serious, with many relapses. *E. H.*

RESSELER (R.). Action thérapeutique de la chrysobiose dans la spirochétose de Dutton chez les souris. [**The Therapeutic Action of Chrysobiose on *Spirochaeta duttoni* in Mice.**]—*Ann. Soc. Belge de Méd. Trop.* 1935. Dec. 31. Vol. 15. No. 4. pp. 541–549.

The description of the action of a new gold compound, Chrysobiose, prepared by the Union Chimique Belge, which whilst considerably less

toxic than solganal and soganal B [see this *Bulletin*, 1932, Vol. 29, p. 563] possesses a similar therapeutic index, 1 : 50, against *S. duttoni* infection in mice. The author recommends its use for the treatment of African tick fever. *E. H.*

VAUCEL (M.). Le séro-diagnostic de Martin et Pettit au Tonkin (résultats de 800 séro-agglutinations). [**Martin and Pettit's Serum Diagnosis at Tonking (the Results of 800 Serum Agglutination Tests).**]—*Bull. Soc. Path. Exot.* 1936. Mar. 11. Vol. 29. No. 3. pp. 251–257.

Although since 1934 only 4 cases of Weil's disease have been diagnosed at the Tonking hospital, the results of serum agglutination tests suggest that infection with *Spirochaeta icterohaemorrhagiae* may be much more common than is generally supposed.

The patients showing characteristic symptoms all agglutinated in dilutions of 1 : 10,000 to 1 : 12,000. 193 patients in which the disease was suspected gave positive agglutinations in 10 cases with average dilutions of 1 : 1,300. 252 sera of patients with undetermined febrile infections gave 7 positive results with average dilutions of 1 : 1,300. 297 sera forwarded for Wassermann tests gave 13 positive results with average dilutions of 1 : 930. 54 sera from cases of pyomyositis gave 12 positive reactions, a result which is considered to suggest that leptospiral infection may play some part in the aetiology of tropical myositis.

The sera of 15 dogs gave uniformly negative results. *E. H.*

MEYER-MAY (J.) & VAUCEL (M.). La spirochétose ictéro-hémorragique est-elle responsable de certaines myosites tropicales. [**Is Spirochaetal Jaundice responsible for Certain Tropical Myosites ?**]—*Bull. Soc. Path. Exot.* 1936. Mar. 11. Vol. 29. No. 3. pp. 257–263.

A detailed account of the results of agglutination tests against *S. icterohaemorrhagie* of the sera of 54 cases of tropical myositis, of which 12 were positive. [See above.] *E. H.*

MARTIN SANCHEZ (Antonio). Las espiroquetosis humanas en las Islas Canarias. Espiroquetos en esputos, orina y sangre. A proposito de quince observaciones. [**Human Spirochaetosis in the Canary Islands.**]—*Medicina Países Calidos.* Madrid. 1936. Mar. Vol. 9. No. 3. pp. 105–124. With 7 figs. [38 refs.]

The author describes and gives brief clinical notes of 14 patients who were found to have spirochaetes either in their sputa, the peripheral blood or the urine, or in more than one of these. Their presence in sputum had been previously noted, but otherwise these cases are claimed to be the first recorded in the Canary Isles. Jaundice was present in 3 only, fever was not found in 5. The most common symptom was pain, in the abdomen or over the liver, varying in degree ; it was complained of in 12 of the 14 ; spirochaetes were found in the circulating blood in 10, in the sputum in 8, in the urinary deposit in 6 ; in all three in one patient, in blood and sputum in 6, in blood and urine in 4. The subjoined table, adapted from the original, shows clearly the combination of symptoms and signs present in each patient, and its reproduction here will render any detailed clinical account unnecessary. The local

rats were found to harbour *Sp. icterohaemorrhagiae* and some of these 14 cases were instances of Weil's disease possibly contracted indirectly from these animals. *H. H. S.*

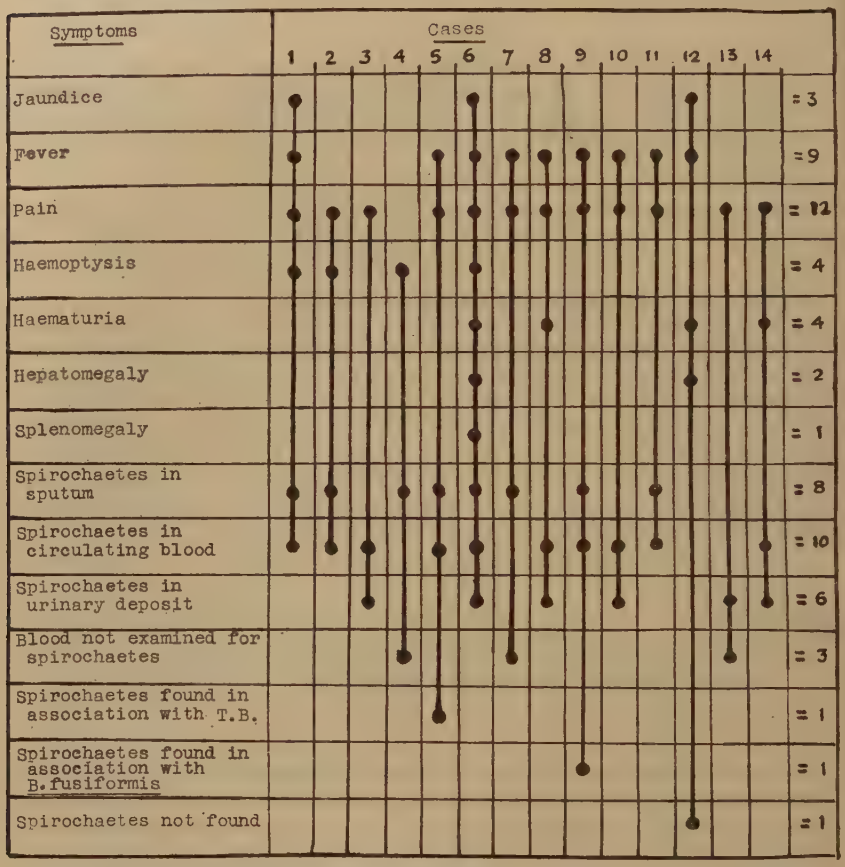


Chart of symptoms of 14 patients with spirochaetosis in the Canary Islands. [Illustrating MARTIN SANCHEZ' paper from *Medicina Países Cálidos*.]

PRETO (Giacomo). A proposito di un bambino portatore di leptospire dell'ittero emorragico. [**Weil's Disease in a Child.**]—*Pathologica.* 1936. Apr. 15. Vol. 28. No. 534. pp. 171–180. With 2 figs. & 1 chart. [30 refs.] English summary (8 lines).

The patient was a boy of 8 years. No history was obtained except that three years before he had suffered from " haemorrhagic nephritis following impetigo " and was cured after two months' treatment. The present illness started two days before he came to the clinic, with fever 39°–40°C., headache, efforts at vomiting, and diarrhoeic stools 8–10 in the day. The liver was enlarged to 2 cm. below the costal margin. The urine was clear, with a slight trace of albumen. Blood culture was negative ; ascaris ova were seen in the faeces. There was a profuse epistaxis on the second day in the clinic ; the temperature fell to normal on the sixth day. There was no jaundice. Examination of the urinary

deposit revealed numerous leptospira. Inoculation into a guineapig resulted in death with the typical findings of Weil's disease. Moreover, the child's serum agglutinated the leptospira markedly in 1 : 100 and to a certain degree in 1 : 300. *H. H. S.*

RAT-BITE FEVER.

HIDAJAT (M. D.) & ESSED (W. F. R.). Een geval van rattebeetziekte, waarbij de prophylactische inspuiting van neosalvarsan gefaald heeft. [**A Case of Rat-Bite Fever. Failure of Neosalvarsan as a Prophylactic.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1936. May 5. Vol. 76. No. 18. pp. 1133–1136. With 3 figs. on 1 plate.

The case recorded is a straightforward one, but worth recording because a fairly large dose of neosalvarsan injected on the day following the bite failed to prevent the onset of symptoms. The patient was a boy of 9 years bitten by a rat on the upper lip during sleep. The following day 200 mgm. of neosalvarsan were injected intravenously, but symptoms came on a fortnight later. Inoculation of the child's blood subcutaneously into mice reproduced the disease. *H. H. S.*

COLE (Arthur F.). **Rat-Bite Fever from a Kitten.**—*Brit. Med. Jl.* 1936. Mar. 28. pp. 638–639.

The description of a typical case of rat-bite fever in a patient who became infected after being scratched and bitten in the finger by a sick kitten. The patient was treated for septic lymphangitis for three months before the nature of the disease was recognized, and confirmed by inoculation of gland tissue into a guineapig. During this period the patient had 10 to 12 attacks of fever, and a profuse development of hair had taken place on the right forearm and dorsum of the hand and fingers. The case was treated successfully with intravenous injections of neokharsivan. *E. Hindle.*

LAVERICK (J. V.). **Rat-Bite Fever from a Cat.**—*Brit. Med. Jl.* 1936. Mar. 28. pp. 639–640.

A similar case to the one recorded above. The patient developed a typical clinical history of rat-bite fever after being severely bitten by a cat. The causative organism was not isolated but the patient was cured by a course of injections of neokharsivan. *E. H.*

BLANC (G.) & NOURY (M.). Infection du merion (*Meriones shawi* Lataste) par le *Spirillum minus* Carter. [**The Infection of a Merion** (*Meriones shawi* **Lataste**) **with** *Spirillum minus* **Carter.**]—*Bull. Soc. Path. Exot.* 1936. Apr. 1. Vol. 29. No. 4. pp. 383–388. With 3 figs. [30 refs.]

The record of a case of spontaneous infection with *Spirillum minus* in a merion, *Meriones shawi*, from Marrakech. The infection was discovered when making passages in guineapigs with the blood of this animal. Only after four passages in guineapigs were spirilla discovered, but the animals in the earlier passages showed pathological changes characteristic of this infection in guineapigs.

The merion, therefore, can be added to the long list of rodents and other animals which may be infected with rat-bite fever. *E. H.*

KNOWLES (R.), DAS GUPTA (B. M.) & SEN (S.). **Natural *Spirillum minus* Infection in White Mice.**—*Indian Med. Gaz.* 1936. Apr. Vol. 71. No. 4. pp. 210–212. With 1 chart & 1 fig.

An examination of 31 white mice originally received from the Central Research Institute, Kasauli, and subsequently kept under conditions precluding the possibility of any rat reaching them resulted in the discovery that 15, or 48 per cent., showed natural infection with *Spirillum minus*. The strain was isolated and shown to be pathogenic to man by inoculating a volunteer who subsequently developed rat-bite fever.

This observation shows that the white mouse may not be a reliable animal for blood inoculation in testing for *Spirillum minus* and the guineapig may be more suitable, for no infection was found in the first 20 individuals examined. *E. H.*

STROESCO (G.). Le mode de transmission et de propagation de la spirochétose provoquée par le *Spirochaeta muris*; son organotropisme. [**The Method of Transmission and Spread of Spirochaetosis caused by *Spirochaeta muris*; its Organotropism.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 7. pp. 619–621. With 2 figs.

An examination of the urino-genital glands of male and female mice infected with *Spirillum minus*, by means of Dieterle's impregnation method, has shown that these organisms have a preference for the vesiculae seminales and Bartholin's glands. The examination of 34 males and 6 females showed massive infections of these organs, up to as many as 100 spirilla in each microscopic field in the case of the older animals. The organisms occur not only in the contents of the vesiculae seminales but also in the glandular cells of the walls. Other parts of the genital system remain negative. In females, Bartholin's glands are always positive and the infection is often present in the periglandular tissue, especially in lactating individuals. During lactation the spirilla leave Bartholin's glands and by means of the blood system reach the mammary glands where they multiply and can infect newborn animals [see this *Bulletin*, 1934, Vol. 31, p. 855]. The infection can also be transmitted during copulation.

In view of the predilection of *S. minus* for these glands, their secretion seems to offer an optimum medium for their culture *in vivo*. *E. H.*

MONTEL (R.) & TRUONG-VAN-QUE. Traitement du sodoku par les sels de bismuth. [**The Treatment of Sodoku by Bismuth Salts.**]—*Bull. Soc. Path. Exot.* 1936. Jan. 8. Vol. 29. No. 1. pp. 33–35.

The authors have treated two cases of this disease by means of injections of Bivatol and both were completely cured after four intramuscular injections, at intervals of 3 to 4 days. A third case, also an Annamite, had been treated by intravenous injections of novarsenobenzol, up to a dose of 0·45 gm., but the course did not prevent a relapse which developed ten days after completion of the arsenical treatment. A course of Muthanol was given, which at once arrested the development and effected a complete cure.

The authors recommend the use of these bismuth salts, in place of arsenicals, as being more convenient and less dangerous than treatment by novarsenobenzol. Moreover they seem to be more efficacious.

E. H.

DYSENTERY.

Amoebic.

Seckinger (D. L.). **The Epidemiology of** *Endamoeba histolytica* **Infection in Two Rural Georgia Counties.**—*Southern Med. Jl.* 1936. May. Vol. 29. No. 5. pp. 472–477. [23 refs.]

The area investigated lies about 50 miles north-east of Atlanta City, in Barrow and Walton Counties. The enquiry started in the town of Bethlehem in which dwell 42 families, all white. Water is obtained entirely from surface wells; sewage disposal consists of open privies, flies are abundant and houses are not screened. Among the 117 residents one active case of dysentery and 13 cyst passers were detected; all but one was discovered at the first examination, so the infection rate is at least 12 per cent. Cases in Bethlehem started after the immigration of two women who had contracted infection outside, the previous year. The area of enquiry was therefore extended. In Barrow County 332 individuals in families giving a history of dysentery were examined, and 66 or 20 per cent. were passing *E. histolytica*, 12 or 8·2 per cent. in vegetative form and the others the cystic. In Walton County 73 were positive out of 189, or 38·6 per cent. Thus in these two counties, and excluding Bethlehem, 129 were positive among 521 persons examined. Nearly one-third, 40, were in the 5–14 year group. In many instances there was more than one infected in a family. In 31 families in Barrow County 74 persons were positive, and 88 in 38 families in Walton County. In one large family of 11 persons, only one was negative, a child of 3 years. Further investigations showed that the family food-handler was at times the source of the spread, but a very potent factor was contamination of food by flies.

H. H. S.

Koiter (D.). Mededeeling uit de practijk. [**Amoebic Dysentery in a Dog.**]—*Nederl. Indische Bladen v. Diergeneesk.* 1936. Apr. Vol. 48. Nos. 1 & 2. pp. 87–89. English summary (5 lines).

A dog in Bandoeng was observed to be passing stools containing blood and mucus. Faecal examination revealed abundant Trichomonas, but no hookworms. A specimen, however, sent to the Pasteur Institute was reported to contain a few ancylostome ova, very many Trichomonas and numerous vegetative forms of Entamoeba. It was treated with emetine hypodermically and by Yatren 125 mgm. and rivanol 10 mgm. by mouth thrice daily and was cured. This is said to be the first certified case of amoebic dysentery in a dog in the Dutch East Indies.

H. H. S.

Wagner (Oskar). Experimentelle Untersuchungen ueber Amöbenruhr. I. Teil. Krankheitsverlauf bei künstlicher und spontaner Uebertragung der Amöbiasis im Versuchstier. [**Course of the Disease in Experimental Amoebic Dysentery.**]—*Beihefte z. Arch. f. Schiffs- u. Trop.-Hyg.* 1935. Vol. 39. No. 1. pp. 1–48 (1–48). With 6 figs. [84 refs.]

Bieling (Richard). Experimentelle Untersuchungen ueber Amöbenruhr. II. Teil. Die experimentell erzeugten Veränderungen

und die Pathogenese der Amöbiasis. [**Pathology of Experimental Amoebic Dysentery.**]—*Ibid.* No. 2. pp. 49–108 (1–60). With 29 figs. [49 refs.]

These two papers describe the results of investigations carried out at Frankfurt on a strain of *Entamoeba histolytica* which was isolated in Hamburg from a case of human amoebiasis by rectal inoculation of a kitten. The strain was maintained for a period of 8 years, during which several thousand cats and several hundred dogs were infected. The work was undertaken primarily with the object of carrying out chemotherapeutic studies on amoebic dysentery, but the very large series of animals used has enabled conclusions to be drawn regarding the susceptibility of animals, the variation in virulence of the amoebic strain, the pathology of experimental infections and a number of other important questions relating to the dysentery amoeba.

The first paper deals with the experimental side of the subject. It is noted that the routine method of infecting animals was by the injection into the colon, through a rubber rectal tube, of saline washings from the large intestine of an infected animal. The washings were obtained by injecting and withdrawing warm saline solution by means of the tube and syringe apparatus. Owing to the failure of some animals to become infected at the first injection, in spite of abundance of amoebae in the material used, it is necessary, in order to avoid losing the strain, to inoculate several animals at each passage. There were very marked differences in the type of infection produced, which varied from the acutest form of dysentery to a purely carrier condition. If the number of animals used had been small it would have been possible to interpret these differences as due to changes in virulence in the strain of amoeba employed. It became clear, however, as the work proceeded that the virulence had remained unchanged and that the differences noted were due to variations in the susceptibility of the animals. Young animals were generally more susceptible than old ones, amongst which occurred cases of infection without symptoms, in which only small amoebae ("minuta" forms) without included red blood corpuscles were to be found. Injection of these "minuta" forms into other cats was capable of giving rise to severe dysentery associated with the presence of large amoebae, with included red blood corpuscles, of the typical *E. histolytica* type. Young dogs were, as a rule, more susceptible to rectal injection than cats, though kittens under 1,000 grams in weight never failed to acquire a fatal infection. With increase in age and weight the susceptibility decreased more rapidly in the case of dogs than in the case of cats. Over a period of 8 years it was noted that the susceptibility of the animals was generally higher during the hot summer months than during the winter, a feature which appears to be correlated with the recognized greater severity of human amoebic infections in hot countries. Such seasonal fluctuations in the susceptibility of the animals emphasize the difficulty there is in attempting to establish distinct species of dysentery producing amoebae, such as *E. dispar*, on biological grounds alone.

Some evidence was obtained that in cats after two or three separate infections a certain degree of immunity was produced. In dogs there was little indication of such an acquired immunity, as these animals appeared to be more liable to repeated infections than cats. Amoebic abscess of the liver, with or without associated bacterial or trichomonas infection, was fairly common in cats but occurred only once in the dog.

Repeatedly it was observed in dogs that the infection of the large intestine induced by rectal injection of infective material spread to the small intestine, while in two instances amoebae were found in the stomach, associated in one of the cases with gastric ulceration. Occasionally dogs, but never cats, became infected accidentally by contact with infected animals. It was found that typical acute amoebiasis could be produced in both cats and dogs by feeding them with amoeba-containing material from such spontaneously infected animals. In general, cats and dogs could be readily infected by introducing cyst-free material from rectally infected cats and dogs into the stomach through an oesophageal tube. In these cases the infections induced were typical ones of the large intestine. Both cats and dogs were infected by administering orally cysts from human cases, but with much longer incubation periods than was the case when amoebae were injected directly from animal to animal.

The second paper deals with the pathology of the experimental infections in cats and dogs, and is illustrated with a number of photographs showing the condition of the intestine and the lesions produced. In a series of 695 cats in which infection followed a single rectal injection one-third showed amoebae in the faeces after an incubation period of two days, while three days later the great majority of the animals were passing amoebae. In a few of the animals there was a longer incubation period, up to 20 days, and in these cases it is noteworthy that when well established the disease was as severe as in animals showing an incubation period of only two days. Except in the case of a few of the larger animals death occurred in a few days. Following both rectal and oral administration of infective material two points of the large intestine are first attacked, namely the lower part of the large intestine immediately above the anal ring and the region just below the ileo-caecal valve. This primary localization applies both to cats and dogs. It is interesting to note that after the administration of certain dyes the staining of the mucosa is most intense at these two points, an indication that it is here that stasis occurs. The first change in the intestine is the appearance of small punctiform haemorrhagic necrotic areas. These gradually extend and become confluent. It would appear that the amoebae which accumulate and even reproduce at the two sites mentioned give rise to a substance injurious to the mucous membrane. The superficial epithelium including the distal part of the crypts is thrown off, leaving exposed both the openings of the crypts and the intervening connective tissue. Amoebae appear to enter the tissues in three ways. They pass directly into the exposed connective tissue, into the crypts or into the opened up lymph channels, whence they migrate to the lymph follicles and the sub-mucosa. Lodged in the tissues or in the crypts, the amoebae multiply and, by virtue of a poison or toxin, produce zones of necrosis which increase in extent and unite with other similiar zones. The intestinal mucosa responds to the invasion by the increased production of mucus which, mixed with blood, forms an excellent medium for the development of amoebae on the surface of the intestine. This mucus, spreading over the mucosa, leads to fresh invasions and ultimately, it may be, to amoebic necrosis of the entire surface of the mucosa of the large intestine. In cats the ileo-caecal valve forms a definite barrier to extension of the invasive process but in dogs this is not so, for infection of the small intestine in varying degree is of common occurrence.

During their development in the tissues the amoebae bring about destruction of lymphatics and capillaries, with the result that amoebae may enter these vessels and make their way to the lymphatic glands and the liver. In sections of the liver the amoebae can sometimes be seen in the capillaries. By multiplication and toxin production a coagulation necrosis of the liver tissue is produced, leading to the characteristic liver abscess. It is noteworthy that though the tissue necrosis evidently results from the local action of a poison or toxin elaborated by the amoebae, it is not very evident that this toxin has more than a purely local effect, for dogs which have had extensive involvement of both the large and small intestine have during the course of some weeks shown no symptoms of generalized toxaemia. Though the course of infection in dogs is very much the same as in cats, in dogs the involvement of the mucosa is generally more superficial, while very characteristic of these animals is the production in the large intestine of distinct ulcers clearly separated from one another by relatively healthy mucosa. In consequence of the more superficial nature of the infection in dogs passage of the amoebae into the blood vessels and production of liver abscess are less commonly observed than in cats. As the disease in dogs is less acute than in cats, spontaneous recovery is more common, though, as in cats, relapses may occur. In spite of the fact that symptoms of general toxaemia are not evident there undoubtedly occurs some absorption of toxins, for a condition of allergy is developed in dogs which have suffered from chronic infections. This is made manifest by the specific skin reaction following injection of amoebic antigen prepared from the heavily infected mucosa of a cat or dog. An emulsion of the mucosa is digested for about 5 weeks with *Bacillus mycoides*. with a view to destroying the proteids. The resulting digest is centrifuged after the addition of formalin 0·02 per cent. when a clear solution having antigenic properties is obtained. The skin reaction takes the form of a rapidly developing nodule surrounded by a red area. In cats it was not possible to obtain the reaction, probably because the disease in these animals is not of sufficient duration. Whether this test and complement fixation will prove to be of use for the diagnosis of human infections future work alone will show. Such are a few of the many interesting points brought out by this extensive and carefully conducted investigation on experimental amoebiasis in animals. The two papers should be carefully studied by all those who contemplate embarking upon similar work. *C. M. Wenyon.*

KUBO (Michio). **Investigations on Amoebic Dysentery. IV. Experimental Studies of Amoebic Dysentery in Dogs. (Second Report.) Amoebic Dysentery of Dogs in Chronic Stadium and the Significance of Dogs in the Spread of this Disease.**—*Jl. Oriental Med.* 1936. Apr. Vol. 24. No. 4. [In Japanese pp. 739–755. With 5 figs. on 1 plate. [45 refs.] [English summary pp. 47–48.]

Examination of 85 street dogs caught in Mukden showed 7 of them to be infected with *E. histolytica* and 2 were cyst-passers. Dogs infected experimentally were examined at intervals of 11 to 40 days after infection, *i.e.*, in what was regarded as the chronic stage [though this appears to have been arbitrarily selected]. Some were then passing typical dysenteric stools containing blood and mucus, others had stools apparently normal but with small quantities of mucus in which the amoebae

could be seen. Post-mortem examination showed, of course, in the acute stage erosions and ulcers; these were sometimes visible in the "chronic" stage, but some showed no macroscopic lesions. Amoebae might be found in the lumen without indications of invasion of the gut wall. *H. H. S.*

CARBONELL Y SALAZAR (A.) & SANCHEZ SANTIAGO (B.). Absceso hepático de una niña de catorce meses. [**Liver Abscess in a Child of Fourteen Months.**] *Archivos Med. Infantil.* 1936. Apr., May–June. Vol. 5. No. 2. pp. 143–151. With 4 figs. [13 refs.] English summary.

The child, a little girl of 14 months, had been suffering, 20 days prior to admission to hospital, from dysenteric symptoms, diarrhoea and passage of blood and mucus with high fever. These symptoms improved but the general condition did not and a swelling in the mid-axillary line at the level of the base of the right lung called for prompt admission. X-ray and puncture with withdrawal of greenish-yellow fetid pus confirmed the suspicion of abscess. The swelling could be completely reduced by steady pressure. Operation was performed and the child recovered. Repeated examination failed to reveal *E. histolytica*; Gram-negative bacilli and Gram-positive diplococci were found, and the abscess appeared to arise via the portal radicles, but the actual causal organism was not determined. *H. H. S.*

MOTTA (Orlando Cabral). Abcesso amebico do pulmão. [**Amoebic Abscess of the Lung.**]—*Brasil-Medico.* 1936. Apr. 4. Vol. 50. No. 14. pp. 294–299. With 4 figs. & 1 chart. [10 refs.]

The patient was a woman of 28 years. Some time before [how long is not known with certainty] she had suffered with dysenteric symptoms —colicky pain, diarrhoea, passage of blood and mucus. When she came under the author's care she presented signs and symptoms of abscess of the right lung, shown to be amoebic. The radiograms showed that the lower part of the lung was not affected; there was a zone of normal lung between the diaphragm and the lower limit of the abscess. Emetine was given by intramuscular injections and also 10 cc. ampoules of 10 per cent. hyposulphite of sodium daily, 20 cc. of 50 per cent. hypertonic glucose twice daily, and neosalvarsan, all intravenously. Death occurred suddenly 23 days after admission. [No operative measures are reported.] From the situation of the abscess the author concludes that the entamoeba did not pass from the liver, *via* the diaphragm, but by the vascular route. If a post-mortem examination was made, there is unfortunately no account of the findings. *H. H. S.*

BASSLER (Anthony). **Intestinal Obstruction due to Amebiasis.**—*Jl. Amer. Med. Assoc.* 1936. June 6. Vol. 106. No. 23. pp. 1965–1968. With 3 figs. [12 refs.]

The patient, a man of 50 years, complained of abdominal cramps and occasional passage of blood after taking laxatives. Sigmoidoscopic

examination revealed inflamed areas and vegetative forms of *E. histolytica* were found. The amoebae disappeared after the patient received chiniofon and he gained 30 lbs. in weight. Cysts were seen occasionally afterwards, and 8 months later symptoms returned and the amoebae were again present. Treatment by carbarsone and later by vioform again was followed by improvement, but a year later he had complete intestinal obstruction with faecal vomiting necessitating urgent operation and an " annular neoplasm " was reported at the rectosigmoid junction. Caecostomy was done and 5 weeks later this wound became inflamed and started to slough and amoebae were found in the edges of the wound. The time from infestation to complete obstruction was 22 months.

Other authors have reported neoplasms of amoebic origin or inflammatory masses secondary to amoebic ulceration, and due to secondary bacterial invasion in many cases. The lesion in the present instance proved to be entirely intra-enteric and cleared up on prolonged anti-amoebic treatment by vioform, emetine, carbarsone and chiniofon by mouth and by enema through the colostomy wound. *H. H. S.*

GOLOB (Meyer). **Oxygenation in Relapsing Amebiasis. Report of Case.**—*Jl. Amer. Med. Assoc.* 1936. May 16. Vol. 106. No. 20. pp. 1725–1726.

Felsen in 1931 advocated a process of intestinal oxygenation for treatment of ulcerative colitis. The patient whose case is recorded here suffered from amoebic dysentery and to all appearances recovered after treatment with emetine and arsenicals. Two and a-half months later the symptoms returned and in a severe degree. Entamoeba cysts were found but no vegetative forms and the condition proved resistant to various methods of treatment and the patient was rapidly going downhill. The author regarded the case as one of " secondary ulcerative colitis, based on a mucosa made vulnerable by the amoeba, an environment in which even normal colonic dwellers, saprophytes, become virulent and pathogenic." Oxygen was administered *per rectum* in the dosage shown in the attached table, during the hours 8 a.m. to 8 p.m., the dosage and frequency being governed by the tolerance of the patient. Castor oil was given at night and seemed to enable the patient to tolerate better the distension caused by the oxygen. Improvement was rapid, the patient leaving hospital on August 17th, less than 3 weeks from the start of the oxygenation. *H. H. S.*

Oxygenation Administered.

Date	Minutes	Bubbles per minute
July 23, 1935	160	20
,, 24 ,, ...	160	20
,, 25 ,, ...	240	40
,, 26 ,, ...	120	20
,, 27 ,, ...	180	20
,, 28 ,, ...	180	20
,, 29 ,, ...	240	20

Total: 21·33 hours ... Approximately $6\frac{1}{8}$ liters.

BACILLARY ; BALANTIDIAL ; GIARDIASIS.

WILSON (D. A. O.). **An Outbreak of Dysentery due to** *B. dysenteriae* **Flexner Type P288.**—*Jl. Roy. Army Med. Corps.* 1936. Mar. Vol. 66. No. 3. pp. 191–192. [Summary appears also in *Bulletin of Hygiene.*]

In June of last year among 43 men of the Indian platoon of the 1st Battalion the K.R.R.C., stationed at Mingaladon [the paper does not state where in India Mingaladon is situated, and the name is not mentioned in the Times Atlas], 9 were attacked with dysentery ; one British soldier was also infected. Six of the cases were mild, but the symptoms were typical, and 4 were of moderate severity. An organism giving the biochemical reactions of *Bact. dysenteriae* Flexner was isolated from all and in 8 of them in almost pure culture. It rendered glucose, mannite and dulcite acid, without gas production. It was, however, not agglutinated by the "classical" Flexner antiserum but with subgroup B antiserum and in low titre (1 : 25) with subgroup A 2 antiserum, with one exception. Subcultures of all the strains were sent to the Meerut laboratory where they were identified as Type P288. BOYD, J. S. K. described it in 1932 [see *Bull. of Hyg.*, 1935, Vol. 10, p. 124] and the evidence in favour of its pathogenicity is strengthened by the facts that it was isolated in every case, was present in many in almost pure culture, and agglutinins against it were found in the serum of one of the patients.

The author states that the source of the outbreak was not discovered, but he makes a fairly shrewd guess in saying that the Indians' latrine was only 6 yards from the door of the cook-house, that the soil outside was probably contaminated by the men washing at a tap after defaecation and that the cooking utensils were cleaned with sand from this area. The outbreak, which occurred from the 4th–16th June, ceased as soon as the practice was stopped. *H. H. S.*

MURASHIMA (Tetsuo). **An Epidemiological Observation of the Dysentery Epidemic which occurred in Kawasaki City.**—*Jl. Public Health Assoc. Japan.* 1935. Sept. Vol. 11. No. 9. pp. 1–5. [Summary appears also in *Bulletin of Hygiene.*]

In January 1935 an explosive outbreak of dysentery occurred in Kawasaki City, a town of 150,375 inhabitants, contiguous to Yokohama. The previous incidence of dysentery was 1·1 per 10,000 and 4·5 for Yokohama. There are two water supplies, a larger at Tode estimated for 100,000 persons and a smaller at Nakahara. Night soil is removed by contractors and used as fertiliser.

The first case was reported on 31st December, 1934 ; thence on successive days 16, 143 and 112, after which notifications fell, but the total by the end of the month was 1,357 or 9·0 per thousand inhabitants. Of the total 32,685 households 1,106 were attacked. In 922 there was one case only, in 135 two, in 40 three, in 7 four, in one there were 5 and in another 11 cases [these total 1,356]. Forty-eight of 67 districts into which the city is divided were attacked. The fatality rate was 22·7 per cent. among 2-year-old children, 19·2 for the 3-year-old, 22·0 for 4-year-old. More than one type of *Bact. dysenteriae* appear to have been concerned, those mentioned are the Shiga, the Nishiura and the Kawasaki organisms, but the latter are not further defined.

In a second section of the paper, the mode of infection of 449 patients is mentioned ; 160 were infected through food, 201 by water, and 88 by contact. Bacteriological examinations of the water sources showed *Bact. coli* to be present in all and " occasionally in the water supplied." Till the autumn of 1934 chlorination of the supply had been carried out but " was discontinued due to the breakage of tools." *H. H. S.*

YANG (Y. N.) & SUNG (C. H.). **A Report of Laboratory Examinations in a Dysentery Epidemic in Kiangsi with Special Reference to the Examination of Carriers.**—*Chinese Med. Jl.* 1936. Feb. Supp. No. 1. pp. 275–279. [Summary appears also in *Bulletin of Hygiene.*]

Since 1928 there have been almost every year outbreaks of dysentery of an epidemic character in Kiangsi Province, and particularly in the districts of Ningtu, Huichang and Hsingkuo. In the summer of 1934 cases numbered thousands and fatalities were many. In the last month of 1934 and the first of 1935 a survey was made of 23 Army hospitals, field and base. There was practically no isolation of sick and wounded soldiers, and almost complete neglect of sanitation.

During the two months 2,674 samples of faeces were examined, only one examination for each patient. The method employed was the lactose fuchsin agar and the China blue rosalinic acid plates for isolation of organisms, followed by agglutination with a Shiga and a mixed Flexner sera. Of the 2,674, 1,069 were from " dysentery patients " and of these 146 or 13·7 per cent. were positive ; 40 were excreting *Bact. dysenteriae* Shiga and 106 *Bact. dysenteriae* Flexner. Among 1,066 ill with other diseases, malaria, enteric fever, etc., 112 or 10·5 per cent. were passing the dysentery bacteria, 42 Shiga and 70 Flexner. Among 277 healthy hospital orderlies 5·4 per cent. (15) were carriers, 3 Shiga, 12 Flexner, while of 262 healthy students 21 or 8·0 per cent. were carriers, 4 of Shiga, 17 of Flexner. Of the total examined, therefore, 294 or 11·0 per cent. were positive ; 89 or 30·2 per cent. of the positive, 3·3 per cent. of the total, were passing the Shiga organism and 205 or 69·8 per cent. of the positive and 7·6 per cent. of the total, one of the Flexner group [in the text the figures are erroneously given as 7 of Shiga to 2·3 Flexner]. Clearly, the condition of the Army hospitals leaves much to be desired and the number of carriers among the apparently healthy is high. *H. H. S.*

NAVOKA (Josaia R.). **Dysentery Epidemic in Ra.**—*Native Med. Practitioner.* Suva. 1935. Sept. Vol. 2. No. 3. pp. 269–272.

This account of an outbreak of dysentery is of more than ordinary interest in that it is contributed by a Native Medical Practitioner qualified from the Suva Medical School. In the first four months of 1935 there were 85 cases admitted, 73 Fijians and 12 Indians, and 16 ended fatally, a case mortality rate of 18·8 per cent. Thirty-seven (14 fatal) were children under 5 years of age, 15 (none fatal) between 5 and 12 years and 33 (2 fatal) above this age. The fatality was higher among the Fijians, 15 out of 73 or 20·5 per cent., but only one among the 12 Indians. Thirty-seven specimens of stools were sent to the Suva Laboratory, and 9 were positive ; in 8 the organism was *Bact. dysenteriae* Shiga.

The steps taken to deal with the outbreak are detailed and show that everything was done that was possible to prevent further spread. The treatment in hospital is detailed. Before being allowed to return to his village, the patient is transferred to the convalescent ward and kept under observation for a week while taking full diet and having no treatment. Only if he remains well during this period is he given his discharge from the hospital. *H. H. S.*

SEKI (K.). **The Clinical Observation of the Ekiri and Dysentery of Children.**—*Oriental Jl. Dis. Infants.* 1935. July. Vol. 18. No. 1. pp. 8–10. [Summary appears also in *Bulletin of Hygiene.*]

Ekiri is the name given in Japan to a disease showing certain points of resemblance to severe dysentery with toxic phenomena. "Clinically," states the author, "it is impossible to discriminate Ekiri . . . from dysentery." The author analyses 382 cases observed by him in 3 years. Children are chiefly attacked, many in the 2–5 years period; it is commonest in the summer, with a maximum prevalence in August. Fifty of the 382 died, a 13 per cent. fatality. Serious symptoms usually came on early, often in 6 hours, at most 36, and on an average 18 hours. The symptoms are feeble pulse, marked cyanosis, convulsions and coma, with diarrhoea and passage of mucus, blood and pus. Death occurred usually within 36 hours, 40 died in 17 hours. In 84 patients convulsions preceded the other symptoms. Rise of temperature occurred at the onset, but disappeared by the 5th or 6th day as a rule and might do so as early as the second day. *H. H. S.*

TEN BOKKEL HUININK (A.). Dysenterie bij kinderen. [**Dysentery in Children.**]—*Nederl. Tijdschr. v. Geneesk.* 1934. Aug. 24. Vol. 79. No. 34. pp. 4051–4056. English summary (9 lines).

An account is given by the author of an illness of varying degree of severity occurring in four children of one family. One died within a short time of becoming ill. The second had symptoms of an acute toxic condition—sunken eyes, torpor and weakness of pulse; the third had one day's elevation of temperature but was scarcely ill, while the fourth had only a slight rise of temperature. All had too frequent stools without a true diarrhoea and the frequency had disappeared in a few days. A slight admixture of blood and mucus was only evident in two of the cases. This series is brought forward along with some other illustrative cases to show how Sonne dysentery, for that bacillus was isolated from the stools, can give rise to serious toxic symptoms or even prove fatal. This is quite a common experience in the Netherlands Indies although the affection is usually regarded as benign in Holland. "Stress is laid on the fact that in Holland in all cases of acute intoxication accompanied by fever, even when no symptoms from the intestinal tract are present, the stool should be examined" for dysentery bacilli. *W. F. Harvey.*

MANSON-BAHR (Philip). **The Differential Diagnosis of Diseases of the Colon (Dysentery and Colitis).**—*Lancet.* 1936. Apr. 4 & 11. pp. 759–765; 830–834. With 12 figs. & 1 coloured plate.

The author very wisely allows himself considerable latitude in his interpretation of "differential diagnosis," and includes in his survey

much valuable information on the aetiology and pathology of the diseases which may give rise to the symptom-complex of dysentery. Thanks to the investigations and teaching of the author and other workers, much of the ground covered is already familiar to those who are concerned with tropical disease.

The author protests against the practice of diagnosing amoebic dysentery on the sole evidence of the presence of Charcot-Leyden crystals, pointing out that he has demonstrated these bodies in cases of intestinal coccidiosis, ulcerative colitis, and carcinoma of the rectum. He agrees, however, that when these crystals are present in large numbers, a prolonged search should be made for the dysentery amoeba. *Giardia lamblia* is considered to be definitely pathogenic, in certain conditions, but the author keeps an open mind as regards the pathogenicity of other intestinal flagellates which, he points out, may be an added infection on top of amoebic or bacillary dysentery. This is true, too, of giardiasis in which condition a search, sometimes of necessity lengthy and repeated, may disclose an underlying amoebic infection. Ulcerative colitis is postulated as a systemic blood dyscrasia due to some unidentified specific infective agent, the primary intestinal lesion being caused by excretion *via* the mucous membrane of toxic products. Another cause of dysenteric symptoms, less generally recognized than the foregoing, is an involvement of the rectum in lymphogranuloma inguinale which results in chronic inflammation and even stricture, a condition especially encountered in China.

These Lettsomian Lectures are comprehensive and essentially practical, and the clear picture presented will be of help in clearing up some of the existing confusion regarding the manifold causes which may produce the dysenteric syndrome. *W. P. MacArthur.*

HETSCH (H.) & ANTON (H.). Der heutige Stand der Lehre von der bazillären Dysenterie und ihrer Bekämpfung.—Reprinted from *Med. Welt.* 1935. No. 12. 10 pp.

TIMPANO (P.) & CASTORINA (G.). La balantidiosi nei bambini. [**Infection of Children by Balantidium.**]—*Pediatria.* 1935. Apr. 1. Vol. 43. No. 4. pp. 449–453. With 4 figs. English summary (2 lines).

From the district of Calabria in Sicily the authors report 4 cases of balantidiosis in children 5 to 9 years of age. Symptoms attributable to the dysenteric condition were present. In all cases a cure was effected by oral administration of arsenical combined with enemata of quinine, ipecacuanha or thymol. *C. M. Wenyon.*

YERED (Domingos). Algumas considerações sobre um caso de balantidiose intestinal e seu tratamento. [**A Case of Balantidiosis.**]—*Brasil-Medico.* 1935. Dec. 7. Vol. 49. No. 49. pp. 1093–1094.

A man, 71 years of age, a pig-breeder had suffered with dysenteric symptoms for over 3 years. He passed 14 or more stools daily, containing blood and mucus and was becoming very weak and emaciated. Examination of the faeces revealed *Balantidium coli* in large numbers, 6–7 per field. A new remedy, Carobinase, was tried ; this is a watery extract of *Jacarandá decurrens* administered, 25 gm. in 500 cc. hot

water, as a rectal lavage. It has been recommended also for Giardia infection, administered by duodenal tube, and by enteroclysis or vaginal douche for Trichomonas. After three weeks the patient's general state had greatly improved and Balantidium was no longer seen. Weekly examinations for five successive weeks continued to yield negative results and the author is convinced that definite cure has been accomplished. *H. H. S.*

BANIK (Lal Mohan). A Case of Dysentery caused by *Balantidium coli*.—*Indian Med. Gaz.* 1935. Oct. Vol. 70. No. 10. p. 566.

JOUCK (N.). **The Pathological Anatomy of Human Balantidiosis.**—*Med. Parasit. & Parasitic Dis.* Moscow. 1935. Vol. 4. No. 3. [In Russian pp. 239–243. With 6 figs.]

An account is given of the histopathology in three cases of human balantidiosis, all of which died shortly after admission into the hospital with symptoms of acute enterocolitis. The pathological changes observed are similar to those previously recorded. *C. A. Hoare.*

ATCHLEY (Floyd O.). **The Maintenance of a Strain of *Balantidium* in Laboratory Rats.**—*Jl. Parasitology.* 1935. June. Vol. 21. No. 3. pp. 183–185.

GLASER (R. W.) & CORIA (N. A.). **The Partial Purification of *Balantidium coli* from Swine.**—*Ibid.* pp. 190–193.

In the first paper the author describes his micropipette method of introducing into a Ringer-serum-rice starch medium single specimens of balantidium from the faeces of the chimpanzee. With the pure-strain culture thus obtained rats were inoculated intracaecally by laparotomy and balantidium infection established. By inoculating the caecal contents intracaecally from rat to rat every three to six days the strain has been maintained for a year.

In the second paper the authors record the culture of balantidia in semi-solid medium in V-shaped tubes. When material is inoculated at the top of one limb the balantidia pass in the course of 24 to 48 hours towards the other limb more rapidly than the contaminating bacteria, so that by repeating the process several times with material pipetted from the other limb the culture can be largely purified of bacteria with the result that in subcultures the ciliates survive longer than they did at first, subculture being only necessary every seven days. *C. M. W.*

NELSON (E. Clifford). **Cultivation and Cross-Infection Experiments with Balantidia from Pig, Chimpanzee, Guinea Pig and *Macacus rhesus*.**—*Amer. Jl. Hyg.* 1935. July. Vol. 22. No. 1. pp. 26–43. With 1 fig. [16 refs.]

Balantidia obtained from the caeca of recently killed pigs and guineapigs and from the faeces of the chimpanzee *Macacus rhesus* were introduced into the stomach of 61 rats which had been prepared for infection by placing them for 5 to 10 days on a 85 per cent. carbohydrate diet. The infection in the rats was controlled by daily

examination of the soft faeces which are passed. The balantidia from the chimpanzee and pig established themselves readily in the rat, one animal retaining the chimpanzee ciliate for 129 days and another the one from the pig for 489 days. No variations in morphology or conjugation cycle resulted from the life in the rat. The attempts to infect rats with *B. simile* of *M. rhesus* and *B. caviae* of the guineapig failed, as also did those to cultivate these ciliates in media which readily gave cultures of the ciliates from chimpanzee and pig. The ciliates in cultures were found to conjugate at a time of food abundance rather than at times of hunger. *C. M. W.*

GABALDON (Arnoldo). *Balantidium coli*: **Quantitative Studies in Experimental Infections and Variations in Infectiousness for Rats.** —*Jl. Parasitology.* 1935. Oct. Vol. 21. No. 5. pp. 386–392.

Strains of balantidia from chimpanzees and pigs were studied in 296 experimental rats. It was noted that the strains varied greatly as regards their power of infecting rats and in the intensity of the infections when these occurred. *C. M. W.*

CORRADETTI (Augusto). *Giardia enterica* (**Grassi, 1881**), **the Correct Name for the** *Giardia* **of Man.**—*Jl. Parasitology.* 1935. Aug. Vol. 21. No. 4. pp. 310–311.

The author argues that as GRASSI in 1881 published a paper with the title "Di un nuovo parassita dell'uomo *Megastoma entericum*" the correct name of the parasite variously known as *Lamblia intestinalis, Giardia intestinalis, Giardia lamblia,* etc., is *Giardia enterica* (Grassi, 1881). *C. M. W.*

CALDER (Royall M.) & RIGDON (R. H.). **Giardia Infestation of Gall Bladder and Intestinal Tract.**—*Amer. Jl. Med. Sci.* 1935. July. Vol. 190. No. 1. pp. 82–88. With 2 figs. [19 refs.]

The case reported is that of a man who died in hospital from persistent diarrhoea after 16 years' illness. Laboratory examination had revealed giardia in the stools and vomit. At autopsy a diffuse enterocolitis was found, with superficial ulceration in the colon. The gall bladder was normal in size and contained a brownish-black secretion. It is stated that "some of this secretion, before the gall bladder was opened, was found to be teeming with *Giardia*" which, however, were not seen in sections. The paper unfortunately does not state exactly how the secretion was obtained from the gall bladder before opening, whether by syringe or by pressure through the duct or whether all possibility of contamination with duodenal contents was avoided. The relationship of the flagellate, whose habitat is the duodenum and upper part of the small intestine, to the lesions of the colon is not clear. *C. M. W.*

DIBOLD (H.). Zur Behandlung der Lambliosis intestinalis. [**Treatment of Infestation by** *Lamblia intestinalis.*]—*Med. Klin.* 1935. June 21. Vol. 31. No. 25. pp. 815–817.

The paper concludes that *Giardia intestinalis* is definitely pathogenic and that its eradication depends as much upon the administration

of drugs which may act directly on the organism as upon the treatment of the special organs—gall bladder, liver, pancreas, intestine—to which the symptoms of any particular case direct attention. *C. M. W.*

CAMPONOVO (Luis E.). Las piretrinas como tratamiento de las parasitosis intestinales. Nota previa. [**Pyretrines in the Treatment of Intestinal Parasitism.**]—*Prensa Méd. Argentina.* 1935. Dec. 4. Vol. 22. No. 49. pp. 2371–2372.

The uses of pyrethrum or pellitory (*Chrysanthemum cinerariaefolium*) have an interesting history. Apart from its known insecticide properties in powder or infusion, some 80 years ago it was used in powder form in the Caucasus for expulsion of Enterobius, and 4 years later the infusion was used *per os* and *per rectum* for Ascaris infestation. In 1924 the combined active principle of the flowers was isolated by STAUDINGER and RUDZICKA. This proved to be toxic to cold-blooded animals, but harmless for man and warm-blooded animals.

The author made trial of this in cases of Lambliasis, some of which had resisted all the usual modes of treatment, stovarsol, treparsol, neosalvarsan in high doses, yatren, methylene blue, naphthalene, and jacaranda. He gave a 0·5 per cent. strength in oil, in doses of 5 mgm. twice daily, by mouth and by rectum. In obstinate cases he gives 60 drops of this solution in oil [? emulsion] in 25 cc. of distilled and sterilized water [why sterile ?]. Of 20 cases treated 12 were negative for periods of examination up to three months, and 8 of them were negative after 4 days' treatment. *H. H. S.*

HEGNER (Robert) & ESKRIDGE (Lydia). **Susceptibility and Resistance of Rats to Infections with Trichomonad Flagellates from Rat and Man.**—*Amer. Jl. Hyg.* 1935. Sept. Vol. 22. No. 2. pp. 307–321.

—— & ——. **Absence of Pathogenicity in Cats infected with** *Trichomonas felis* **from Cats and** *Trichomonas hominis* **from Man.**—*Ibid.* pp. 322–325.

Trichomonas from kittens infected experimentally with *T. hominis* or *T. felis* did not support, either from clinical symptoms or post mortem appearances, the view that these trichomonads can be pathogenic to kittens. It was shown that clean cats associating with infected ones might contract an infection. It was also proved that carbarsone was effective in eliminating trichomonas infection from cats.

C. M. W.

CICCHITTO (Angelo M.). Dissenterie miste da *Entamoeba coli-Blastocystis jalinus* e da *Blastocystis jalinus-Trichomonas intestinalis.*—[**The Effect of Blastocystis in Conjunction with Protozoa in Production of Dysenteric Symptoms.**]—*Policlinico.* Sez. Prat. 1935. Aug. 26. Vol. 42. No. 34. pp. 1671–1679. With 2 figs. [41 refs.]

Writing of *Blastocystis* in the human intestine the author expresses the opinion that this organism, harmless when alone, may produce a type of dysentery when associated with *Entamoeba coli* or *Trichomonas hominis.* *C. M. W.*

GABALDON (Arnoldo). **Carbarsone: its Action on *Trichomonas hominis* and on Rat Trichomonads in Vitro.**—*Amer. Jl. Hyg.* 1935. Sept. Vol. 22. No. 2. pp. 326–338. With 2 charts.

Carbarsone diluted in culture fluid at a concentration of 0·3 per cent. was lethal to both *Trichomonas hominis* and a rat trichomonad in less than 72 hours. In weak solutions (0·05 per cent.) the drug retarded the development of the flagellates in culture. *C. M. W.*

MARMO (Achille) Contributo allo studio della metadissenteria.—*Ann. di Med. Nav. e Colon.* 1935. Sept.–Oct. 41st Year. Vol. 2. No. 3–4. pp. 611–651. With 4 figs. [100 refs.]

MISCELLANEOUS.

CLEMENTS (F. W.). **A Medical Survey in Papua : Report of the First Expedition by the School of Public Health and Tropical Medicine to Papua, 1935.**—*Med. Jl. Australia.* 1936. Apr. 4. 23rd Year. Vol. 1. No. 14. pp. 451–463. With 10 figs. & 2 maps. [12 refs.]

In 1912–13 BREINL made a survey of the coastal belt of Papua and described the forms of disease he found [see this *Bulletin*, 1914, Vol. 4, p. 407]. The work undertaken by the present expedition was a village to village survey of an area in the western portion of the Central Division of Papua, the object being " to obtain factors of health and disease amongst a virile vigorous people living in a well-populated area." In two months 400 miles were covered and over 5,000 natives examined. These belonged to three groups, the Roro, a coastal tribe, the Mekeo, a subcoastal, and mountain groups living at 5,000–7,000 feet. The results are set out in tables giving in detail and in summary the vital statistics and disease incidence in each of the three groups. It is seen at once that yaws and two forms of tinea preponderate in the Mekeo and Roro tribes. *Yaws*, the author believes, was introduced from Polynesia in the first half of last century. It has made little progress inland possibly owing to altitude but more probably to the absence of intimate contact with the disease, for when coastal natives were entertained for the first time at a mountain dance several of the mountaineers contracted a severe form of yaws. A table gives the age distribution of 397 cases and demonstrates the usual proponderance of secondary cases in children. The tertiary manifestations included periostitis of the tibia, extensive cutaneous ulceration and scarring, periarticular nodules and plantar yaws.

True *tropical ulcer* was seen in less than 1 per cent. and mostly in young children, none in the mountain districts. *Tinea imbricata* was widely spread in the coastal tribes in both young and old, but some natives seem to have an absolute immunity. *Tinea flava* was not seen in children under 12 years of age.

In the Mekeo the maximum amount of *malaria* occurs in children from 2–5 years ; all children have enlarged spleens ; it is a hyperendemic malarious area (CHRISTOPHERS). The parasite is *P. falciparum* and the vector *A. punctulatus*, var. *mollucensis*. The salting process, it is stated, kills few babies and the Mekeo population is increasing. Two cases of *filariasis* were seen in one village where a night survey showed embryos in the blood of all natives over 45. A *hookworm* survey of one of the Mekeo villages failed to find ova in the stools of the children. The village pigs promptly remove human excreta and it is suggested that this and a good diet keep hookworm disease subclinical. Details are given of seven cases of *leprosy*, believed to be a disease of recent introduction. All were chronic and there were no skin lesions ; it is doubtful whether they were infectious. Of general diseases those seen and discussed are : goitre, infantile paralysis, dermatitis *ab igne*, paralysis agitans, rheumatic carditis, and myasthenia gravis.

Goitre was found in four mountain villages and its aetiology is discussed. The native belief that it occurs in women who eat pig during gestation recalls the statement of MCCARRISON that excessive diets of protein and fat can produce goitre ; feasts of pig in this region may last a month.

The *dermatitis ab igne* is produced by the crouching of the naked natives over a fire, especially in the long cold evenings of the wet season. The anterior surface of the legs is affected.

Chest lesions were noted in 42 natives, all over 30 years. Sputa were examined but T.B. not found and it is believed that these were cases of *chronic bronchitis*. *Tuberculosis* is, however, mentioned elsewhere in the paper.

Tables give death and birth rates in the several villages of the Mekeo and Roro for a quinquennium and the vital statistics of nine British Colonies are given for comparison. [When such figures are given for Nigeria, Nyasaland, British Guiana, etc., it would be well to state that they cover only a small part of the area in question.] In both tribes there is an annual increase, 2·1 and 8·9 per mille respectively. An endeavour to ascertain the causes of death led to the conclusion that common causes are complications of pregnancy and snake bite. Of native customs of medical interest one may cite the following: after the marriage ceremony the bride is excused all manual labour and is fed on nourishing foods till after the birth of the first baby. This had its origin in the hope thereby to prevent miscarriage. Whether the intended effect followed is not stated. *A. G. Bagshawe.*

HOOPER (Robert C. D.) & LOEWENTHAL (L. J. A.). **A Survey of Health Work in Teso, Uganda.**—*Ann. Trop. Med. & Parasit.* 1936. Apr. 8. Vol. 30. No. 1. pp. 17–32. With 1 map & 4 figs. on 1 plate.

This paper is an interesting account of the attempts of an embryonic health service to ameliorate the health conditions of a primitive population with, from the European standpoint, very inadequate resources.

The word "survey" used in the title of the paper has a rather specialized meaning in health literature. This is not a health survey of that kind. No mention is made of birth rates, death rates or morbidity statistics. The difficulties attendant upon public health work among backward communities are, however, graphically described. These are familiar enough to workers in the tropics but are likely to surprise those whose experience has been confined to countries with an older western civilization.

The District of Teso is the northern part of the Eastern Province of Uganda. It has an area of 4,052 square miles, a native population of 300,000, and a European medical and sanitary staff of three—a District Medical Officer, a Nursing Sister and a Sanitary Inspector. A considerable amount of the time of the two former is obviously devoted to curative medicine. Apart from Soroti, the administrative headquarters, there are eleven townships. These consist of single rows of trading premises and dwellings, on either side of a road, and scattered native dwellings usually within a radius of a mile, or a mile and a half, from the market. The standard of living even here is very low. A village may comprise dwellings scattered over an area of five square miles. Adequate inspection of such a village may entail three or four days' walking. Communications are good; most native dwellings are within seven miles of a road or track passable to motor cars. Sanitary control of the movements of the native population is, however, impossible; the African often travels by bush paths or by canoe on lakes.

In the native dwellings the most elementary rules of hygiene are conspicuously ignored. Windows or open ventilation of any kind are

often "tabu," as possible means of access for the evil eye, mosquitoes or other unwelcome visitor.

Plague, dysentery, smallpox and cerebrospinal fever are prevalent from time to time. Yaws, helminthiasis and deficiency diseases constantly affect a proportion of the population. Every adult native has developed a tolerance to the malaria parasite. Trachoma, venereal disease and tropical ulcer are very prevalent.

There are four African sanitary orderlies in charge of the health work of rural areas. Each is posted to the headquarters of a "county" where he lives in a model compound. The average monthly cycle running of these men is 800 miles.

The above excerpts from an interesting paper are sufficient to illustrate the magnitude of the problems facing a small enthusiastic band of health workers. *Norman White.*

MAURITIUS. **Annual Report of the Bacteriological Laboratory for the Year 1935** [ADAMS (A. R. D.), Senior Pathologist & Superintendent]. —22 pp. 1935. Dec. 2.

The Annual Report of the Government Pathologist, Mauritius, again contains much of interest. The use of McCartney's procedure and of "canned media" had considerably facilitated the routine work. The actual number of specimens dealt with in routine was a little less than in the preceding year. These are divided into pathological, including microscopical examinations of blood, faeces, urines, sputa, throat and nose swabs, and tissues, and serological work; bacteriological examination of the same materials and of water supplies; medico-legal; research and miscellaneous. The results of the water analyses are satisfactory in the colder months, less so in the warmer on account of algal growth. Dr. Adams is of opinion that some physico-chemical precipitation treatment prior to filtration would be better than chlorination.

Trypanosoma boylei appears to be common in the *Triatoma rubrofasciata*, the reduviid bug of Mauritius. Dr. MADGE is collecting material from cases of human myiasis; further instances of infestation by *Bertiella studeri* have been observed, 5 having now been recorded in Mauritius.

Two special problems of research have been pursued; the first was the investigation of the molluscan host of the *Schistosoma haematobium* endemic in the island, viz., *Bulinus* (*Pyrgophysa*) *forskali*. This is widely distributed in running water throughout the Colony and has been found on watercress and other edible plants in the market stalls and is, consequently, of much public health importance. Its small size results in its producing only a few cercariae each, but this is compensated by their large aggregations. Previous lack of success in experimental work is ascribed to under-infestation. A survey of the waterways of the island is to be undertaken to determine the distribution of these snails and also a survey of the prevalence of the disease they convey. A second important research problem taken in hand was trypanosomiasis of stock, three species, *T. evansi*, *T. vivax* and *T. theileri*, being found in indigenous cattle. This is dealt with elsewhere in this *Bulletin* [see p. 650].

Four papers have been published by Dr. Adams during the year, namely :—

1. Studies on Bilharzia in Mauritius. II. The Recovery of Adult *Schistosoma haematobium* after Development in *Bulinus* (*Pyrgophysa*) *forskali*. *Ann. Trop. Med. & Parasit.* 1935, Vol. 29, p. 255.
2. Trypanosomiasis of Stock in Mauritius. I. *Trypanosoma vivax*, a Parasite of Local Stock. *Ibid.* p. 1.
3. Ascariasis of the Liver. *Trans. Roy. Soc. Trop. Med. & Hyg.* 1935, Vol. 28, p. 419.
4. A Fourth Case of Human Infestation with *Bertiella studeri* (Cestoda) in Mauritius. *Ibid.* Vol. 29, p. 361.

H. H. S.

VISCHER (Mattheus). Medizinische Erfahrungen unter den Dajaken in Süd-Borneo. [**Medical Practice among the Dayaks of South Borneo.**]—*Schweiz. Med. Woch.* 1936. Mar. 28. Vol. 66. No. 13. pp. 315–320. With 12 figs. [12 refs.]

This is an account by a Swiss missionary of the conditions of life, vital statistics and diseases seen in six years at a mission hospital in S. Borneo. It differs little from similar accounts of primitive natives in other parts of the tropics but comes from a little known region.

These Dayaks inhabit a lowland district " five motor-boat hours " from the capital, Banjermassin, situated on the south coast of the island. The author saw 17,258 patients, representing he thinks 8,000 Dayaks. The population is scattered.

Of tropical disease *malaria* is general, chiefly tertian but also subtertian. Severe forms are rare and blackwater has not been seen. In 1933 malaria formed 26 per cent. of diagnoses. Quinine, plasmoquine and atebrin are all used in treatment. Atebrin as a patent medicine is obtainable by anyone. The author says it has overcome the mistrust of the natives and now threatens the flourishing quinine industry of the Netherlands Indies. Refractory cases, however, are still met with. *Amoebiasis* is said to cause 10 per cent. of admissions. It is treated by emetine and to a greater extent by yatren which is readily obtained by the natives. Spirocid is also useful but the author has seen toxic symptoms follow a moderate dose. Liver abscess is not uncommon ; emetine injections are curative and operation is never needed ; other forms of dysentery are not mentioned. *Yaws* is widely spread. Practically every Dayak suffers or has suffered. Neosalvarsan is the best drug, 0·3 gm. for a grown man ; larger doses may be followed by unpleasant symptoms. Bismuth injections are less effective. Spirocid is useful. The author finds that spirocid and salvarsan are less dangerous in yaws than in syphilis. Syphilis is rare in this region and he sees no objection to spirocid treatment of yaws by school teachers. Owing to the high price and the indolence of the natives there is little risk of overdose. Salvarsan-resistant cases are treated by " Terpin Stahl A," a Swiss preparation recommended by Albert SCHWEITZER. It is estimated that one per mille of the population suffers from *leprosy*. Lepers are segregated by the natives when the disease is far advanced. It is children who contract leprosy, adults very rarely. Of leper colonies he aptly writes—As philanthropic establishments they are indispensable, as research institutions they are important, for the combatting of endemic leprosy they are useless (untauglich). Of other diseases *tuberculosis*, chiefly pulmonary, is wide-spread. *Xerophthalmia* forms 0·5 to 1 per cent. of diagnoses and is found in children between 4 and 15 years, as a rule convalescents from dysentery and

gastro-enteritis. Cod liver oil is rapidly curative. Typical signs of *rickets* were seen in 1 to 1·5 per cent. of treated children; sick children are confined by the natives to their dark houses and fed on rice. *Tinea imbricans* is widely spread and refractory to treatment. Syphilis is very rare and gonorrhoea very mild. A table follows of the frequency with which a number of diseases were seen. The illustrations depict chiefly yaws.

A. G. Bagshawe.

BRUNELLI (Piero). Cenni nosografici sulla regione di Gedda. [**Disease in Jedda and its Environs.**]—*Arch. Ital. Sci. Med. Colon. e. Parassit.* 1936. Jan. Vol. 17. No. 1. pp. 34–48. With 8 figs.

Included in the sanitary and medical organization of Jedda is an English doctor, a Dutch surgery in charge of a Javanese medical man, a Russian Legation Dispensary, and an Italian dispensary, while the local Government entrusts the sanitary measures to the hands of Syrian doctors and pharmacists, and the laboratory is directed by a Turkish doctor. Unfortunately, this article gives no figures, resting content with general vague terms. At the time of the Mecca pilgrimage "tens of thousands" of persons of all ages and either sex are gathered there, young children, old people, many suffering from serious disease, women in advanced pregnancy. Reference is made to the climate, water supply (or lack of it), and prevalence of mosquitoes. The splenic index in the town is very high; it is given as 86 per cent. of the children; of 70 blood specimens positive for malaria 44 showed *P. vivax*, 16 *P. falciparum*, 9 both these, and one *P. malariae* (there was a little uncertainty about this). *Phlebotomus papatasii* abounds, also in the heat of summer *Aedes aegypti*, and many cases of dengue occur. Dracunculus is seen among the Beduins, and a few cases of filariasis, but only in those from the interior. Syphilis is not very common; more frequent are cases of soft chancre and gonorrhoea, and more rarely granuloma venereum [no mention is made of Lymphogranuloma inguinale]. Amoebic dysentery is common and, in children, diarrhoea associated with Giardia. Of helminthic infestations, ascariasis heads the list, ankylostomiasis coming next; one case of *T. saginata* is noted. Trachoma affects "hundreds of victims" every year. Asthma is widespread [nothing is said of the prevalence of tuberculosis].

H. H. S.

WATSON (Alexander J.). **Vincent's Disease.**—*Chinese Med. Jl.* 1936. Jan. Vol. 50. No. 1. pp. 46–49. With 2 charts.

During the four months November 1935–February 1936 the author saw 20 cases of Vincent's angina, occurring in a local outbreak at Yunnanfu, and other milder cases probably escaped observation. The symptoms were, except for difference of degree, very similar in all—thick exudate on the tongue ("half the thickness of the tongue"), difficulty in moving the tongue, some salivation and dysphagia and a deep sore throat, about the level of the hyoid. *B. fusiformis* and spirilla were present, sometimes one predominating, sometimes the other, and there was usually a stomatitis with ulceration of palate, tongue or lips. The false membrane which some writers have reported in cases of Vincent's disease was not seen in those of the author. The temperature was not very high, up to 102°F. or a little higher (102·4°F.).

In some, 2–3 weeks elapsed before free movement of the tongue was regained, the inflammation at the base being severe. *C. diphtheriae* though looked for was not found. *H. H. S.*

SNIJDERS (E. P.). Over het onderzoek van het scleroma respiratorium (rhinoscleroom) in Ned-Indië. [**Investigations into Rhinoscleroma in Netherlands India.**]—Reprinted from *Feestbundel 1936 v. h. Geneesk. Tijdschr. v. Nederl.-Indië.* pp. 397–410.

This is a review of our knowledge of rhinoscleroma and a recapitulation of the steps by which it was established as a definite disease in the Netherlands Indies. A description of the condition is given as: An inflammatory process into which there enter degenerative, exudative and proliferative phenomena. The infiltration is mainly of plasma cells, often degenerated and hyaline, with a variable accompaniment of lymphocytes and polymorphonuclears. It is a chronic condition with very special localization and morphological character. Foamy macrophage cells, sometimes filled with capsule bacteria, may also be a microscopic feature.

Other characters of the disease are that it has been found especially in the Batak lands of Sumatra, that it is endemic, chronically but comparatively slightly infective, and of familial incidence. It is in some respects reminiscent of leprosy and is essentially a disease of "the great unwashed." Evidence of a circumstantial character has been produced that it is a disease of ancient times, still clinging to the Mohammedan element of the ancient Batak people but obviously a disease which is dying out with the advent of hygiene. In nearer British India the disease has been signalled in Chota Nagpur and an endeavour is made to link up the people of this plateau, of the Munda and Kolar stem, with the people of the Batak lands. Research in the Chota Nagpur region might offer some further proof of this connexion.

A typical scleroma is not known in animals nor has the condition been transmitted experimentally to animals, but the author considers that there is a possibility of the pig being a carrier. Pure cultures of what is presumably the causal organism, *Klebsiella rhinoscleromatis*, are obtainable from the deeper tissues of the lesions produced and there is every reason, from cultural and serological tests, with antigen analysis, to regard this organism as specific. It is to be distinguished from *Kl. ozaenae* and *Kl. pneumoniae.* Although these organisms may be mixed with it in nasal mucus, they are not to be found in the deeper parts of affected tissue which, nevertheless, yield pure cultures of *Kl. rhinoscleromatis.* *W. F. Harvey.*

KARUNARATNE (W. A. E.). **The Pathology of Rhinosporidiosis.**—*Jl. Path. & Bact.* 1936. Jan. Vol. 42. No. 1. pp. 193–202. With 26 figs. (4 coloured) on 7 plates. [37 refs.]

"Summary of the fifty-three reported cases.

"*Geographical distribution.* This is remarkable for its occurrence over a wide area and the cases have also been very isolated. Most of the patients have been Indians (40), with a few in Ceylon (4), North America (5), South America (3) and Italy (1). The very wide distribution of the disease is noteworthy. In the solitary European case (Orlandi), a woman, aged 56, developed a rhinosporidial growth in the conjunctiva following injury of the eye by a splinter. Seeber's original

case was also a native of Italy, but he had been resident in South America since his infancy. The case described by Ashworth was that of an Indian student who had come to Edinburgh for purposes of study, and the two other cases referred to in his monograph were also Indians. Of the four cases reported from Ceylon, three had not been out of the island. With my 34 cases, the total number from Ceylon is only a little short of the total of Indian cases. All 5 cases reported from North America occurred in widely separated areas. Only one had been outside the United States and that was for a three days' visit to Canada. The case described by Wright (1907) had lived all his life in the neighbourhood of Memphis (Tennessee), whilst Graham's case, a 12 year old negro boy born in Georgia had resided in a small coal-mining village in Alabama since he was four.

"*Age.* The ages, recorded in 33 cases only, ranged from 8 to 60 years. The disease mainly affects young subjects, 24 of the 33 being below the age of 30 and 12 below the age of 20.

"*Sex.* Only 4 of the 53 cases were females.

"*Anatomical Distribution.* In 40 of the 53 cases the lesion was in the nose, mainly the anterior part. The conjunctiva was affected in 8, the lachrymal sac in 2 cases, and the disease occurred once each in the penis, uvula and external auditory meatus. One patient, a woman, had both a nasal growth and an independent growth on the posterior pillar of the fauces. In my own series of 34 cases, two were conjunctival, one lachrymal and the rest nasal."

POLIDORI (Tommaso). I micetomi in Somalia. [**Mycetoma in Italian Somaliland.**]—*Arch. Ital. Sci. Med. Colon e Parassit.* 1936. Jan. Vol. 17. No. 1. pp. 19–33. With 7 figs.

The author has collected and reports in tabular form cases of Madura foot and other mycotic affections seen by him at the Civil Hospital at Mogadiscio [Mogdishu, on the Benadir coast]. Five of the former and 57 of other forms are mentioned. Many, in fact the majority, are not seen until the later stages, the patients regarding the condition as syphilitic or as resulting from some insect bite until the tumour is of a size to impede walking, or the ulceration, abundance of secretion and foul odour force them to seek assistance. Large doses of potassium iodide rarely give satisfactory results, and in many cases amputation is necessary, though, if the patient is seen early enough, enucleation may succeed. The author has never observed metastases, but if enucleation is not thorough recurrence takes place. All the patients were in a state of failing health, with anaemia, and many of them showed signs of food and vitamin deficiency. *H. H. S.*

PARISE (Nicola). Osservazioni cliniche e ricerche sperimentali sulla nocardiasi.—*Giorn. Ital. di Malat. Esot. e Trop.* 1935. Aug. 31, Sept. 30 & Oct. 30. Vol. 8. Nos. 8, 9 & 10. pp. 195–8, 201–4, 207–8; 234–6, 239; 248, 251–5, 257–8. With 15 figs.

CHOPRA (R. N.) & CHAUDHURI (R. N.). **The So-called Mystery Disease of Calcutta** (*Jhin-Jhinia* **or** *Thartharia*).—*Indian Med. Gaz.* 1936. Apr. Vol. 71. No. 4. pp. 205–209.

This so-called "mystery disease" seems to have started suddenly last September in villages at Bolbule and Katakhali on the Ichamati river and after visiting a village for about a week passed on to the next. As many as 40–50 persons might be attacked in a night. Most were

between 18 and 35 years, Mohammedan females almost entirely at first, latterly both males and females and Hindus also, and most attacks started in the late afternoon. The following is the recorded train of symptoms :—

"The 'disease' is said usually to start suddenly with a tingling sensation in one or both feet, especially the big toes. The sensation then spreads up along the body to the head. Many patients are said to complain also of headache, giddiness and a hot burning sensation in the head. Simultaneously with or soon after this subjective sensation, the victim begins to shiver and may have severe movements of the whole body. The eyes are said to be intensely congested and the body stiff during an attack. Symptoms vary in severity ; they may be mild or severe ; consciousness is as a rule retained. The attack lasts from a few minutes to a few hours."

The name *jhinjhinia* implies tingling sensations, and *thartharia* shivering. Brief details, but all the essentials, are given of 17 cases. The local lay treatment consisted in tying the patient to a tree to keep him erect, as lying down might lead to a fatal rush of blood to the brain ; then cold water, bucketful after bucketful to the number of a 100 or more, is poured over the head to drive the blood back to the legs until the patient asks for a cessation of treatment. The only conditions revealed by physical examination have been an increase in the knee-jerks and in the pressure of the cerebrospinal fluid in some cases. The latter may be accounted for by the effect of the chilling resulting from the water treatment.

The condition is probably a neurosis of the auto- or hetero-suggested category [comparable with latah, tarantism] for considerable notoriety had been given to it in the press, all the patients had discussed it with their neighbours prior to being attacked and a sort of panic concerning it had been set up. *H. H. S.*

SOEGIRI. Een geval van mimi-vergiftiging. [**A Case of King Crab Poisoning.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1936. Apr. 7. Vol. 76. No. 14. pp. 880–883. With 4 figs. on 2 plates. [Summary appears also in *Bulletin of Hygiene.*]

A description is given of presumed mimi or king crab poisoning with fatal result. The patient, a fisherman of powerful build, partook of a meal of this crab in spite of the warnings of his companions. That this was dangerous seems to have been known, but it was also suggested that the danger could be eliminated by a certain mode of cooking. No symptoms followed immediately on the meal, but the man on return with his fellows to their fishing vessel had fallen down suddenly and lost consciousness. The jaws were rigidly fixed open and there was foam upon the lips. No twitching and no vomiting occurred. Death took place about 6 hours after the meal. An autopsy was carried out, which showed no obvious cause of death and in the stomach contents were the remains of a meal of king crab. There is no reason to suppose that death was due to any other cause than that assigned.

W. F. Harvey.

FLECKER (H.). **Cone Shell Mollusc Poisoning, with Report of a Fatal Case.**—*Med. Jl. Australia.* 1936. Apr. 4. 23rd Year. Vol. 1. No. 14. pp. 464–466. With 2 figs.

Fatal results from *Conus* poisoning are rare. The author gives summaries of the symptoms in 5 non-fatal cases referred to by Professor

CLELAND in the *Australasian Medical Gazette*, 1912. Three years later a fatal case was recorded. The present instance was reported in response to a questionary sent to all practitioners in North Queensland asking for information on injuries caused by plants or animals in tropical Queensland.

A man of 27 years picked up a live cone shell (*Conus geographus*) on Haymen Island. He was stung in the palm, a small puncture mark being seen. Local numbness started, without any pain; ten minutes later he felt a stiffness of the lips, ten minutes later still the sight was blurred and there was diplopia; paralysis of the legs set in half an hour later after the stinging and in one hour he lost consciousness and died five hours from the time of the injury. Post-mortem revealed nothing abnormal; the organs all appeared to be healthy. The species in the five cases recorded earlier were *C. aulicus*, *C. tulipa*, both producing mild symptoms, *C. textile* mild, *C. geographus* (2), one certainly severe, and the previous fatal case was due to *C. textile*. In all of these the pain was severe and in one case the patient "writhed in agony." Medical men generally are unaware of the poisonous effects of these animals and the danger of picking up living cone shells, but the natives are fully acquainted with it and treat it with dread and respect.

H. H. S.

BODEAU. À propos de quelques accidents dus au mancenillier. [**Accidents due to** *Hippomane mancinella*.]—*Arch. Méd. et Pharm. Nav.* 1936. Jan.–Feb.–Mar. Vol. 126. No. 1. pp. 122–133.

Hippomane mancinella, the Manchineel Tree, is one of the Euphorbiaceae. It is common in the Antilles and on the shores of the Gulf of Mexico. The fruit resembles a small reddish apple; it has an acrid and rather disagreeable taste, but some persons, sailors in particular, have been known to eat several. Its effect is to produce severe burning pain in the mouth and stomach, followed by nausea and vomiting, and later difficulty in swallowing. The lips and buccal mucosa become swollen and blistered, and eroded aphthous patches appear. There are signs and symptoms of collapse, and death has been recorded.

Apart from the fruit a latex oozes from bruised leaves or broken branches and has an irritant and vesicant action. The effect is often seen on the skin of those who take shelter under these trees from a storm. Small vesicles from the size of a pin's head to a nut, by confluence, are caused. Treatment is purely symptomatic.

The latex has been used as a rubefacient and counter irritant and also as an anthelmintic [but no details as to results are known to the reviewer].

H. H. S.

RAYMOND (W. D.). **The Poisonous Effects of Some Local Species of** *Euphorbia*.—*East African Med. Jl.* 1936. Mar. Vol. 12. No. 12. pp. 369–374.

Nine species of Euphorbiaceae are mentioned by the author. The poisonous properties are not equally potent in all. The latex acts as a local irritant when applied externally and, as would be expected, acts similarly but more severely on the mucous membranes. Thus, if introduced into the eye, it sets up conjunctivitis and even keratitis, ending in blindness; in the nose, sneezing, inflammation and epistaxis; in the alimentary tract, vomiting, diarrhoea, colic, followed by collapse,

convulsions and death. Its action in man differs from that in animals in degree and perhaps in kind. Many plant-eating animals can eat it without harm, and the toxic dose, of *E. resinifera* for example, is for dogs about 30 times as great as that for man. At autopsy, if death has occurred from ingestion of the poison, there are signs of acute gastro-enteritis, ulceration and perhaps perforation.

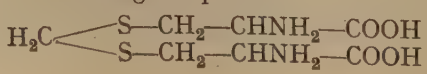
The juice of some varieties is used as an emetic in cases of snakebite, others as an anthelmintic and for gonorrhoea ; the roots and leaves of others as purgatives. [Several members of the Euphorbiaceae are employed for manufacturing arrow poisons, and these act both locally as an irritant and also as cardiac poisons. The Manchineel Tree (*Hippomane mancinella*) a native of the West Indies and Tropical America belongs to the same order. It has a fruit resembling an apple and causes severe burning pain and tenderness in the mouth and stomach, nausea and vomiting, followed by swelling and blistering of the lips and tongue and later by aphthous erosions. See preceding abstract. *H. H. S.*

TONKING (H. D.). **Pyrethrum Dermatitis in Kenya. With Note by** J. H. SEQUEIRA.—*East African Med. Jl.* 1936. Apr. Vol. 13. No. 1. pp. 7–14. [11 refs.]

Pyrethrum, *Chrysanthemum cinerariaefolium*, of value as an insecticide, grows well in Kenya, particularly at a height of 5,000–7,000 feet, and flowers throughout the year ; in fact its cultivation is a growing industry. Probably some 200 Europeans in the Colony are now cultivating it for commercial purposes. The pyrethrum content is 1–2 per cent., as in other countries, *e.g.*, Dalmatia and Japan, but these last have but a short season of growth. Sweating facilitates solution of the irritant constituents and so enhances their action. The effects produced are : Itching at the corners of the eyes, lachrymation, irritation of the nasal mucosa with rhinorrhoea, itching on exposed parts of the body, a papular and vesicular rash, flaking or peeling of the skin and painful fissures. Exposure to sunlight exacerbates the lesions. Some persons show merely a local dermatitis from contact, others systemic allergy. Both Europeans and natives are affected, but as a rule the former more severely. Brief details of six cases are given. Removal from contact and the application of some simple protective ointment suffice for the local treatment, allergic patients must forgo contact with the plant, which probably entails leaving the district, or desensitization methods may be tried. *H. H. S.*

HIJMAN (A. J.) & VAN VEEN (A. G.). Over het djengkolzuur, een nieuw zwavelhoudend aminozuur. [**Djenkol Acid, a New Sulphur-containing Aminoacid.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1936. Apr. 7. Vol. 76. No. 14. pp. 840–859. With 4 charts. English summary. [14 refs.]

Djenkol acid is believed to be responsible for the toxic symptoms occurring after ingestion of the djenkol bean, *Pithecolobium lobatum.* These symptoms are chiefly urinary—pain in the kidney area, dysuria, often anuria ; the urine contains blood and casts and sharp acicular crystals with the following composition



Their presence in large numbers in the urethra may cause necrosis, fistula and extravasation. The djkenol bean has a high vitamin B_1 content and, in spite of its toxic properties, is used considerably as a food. Experiments on rabbits showed that it was oxidized in the body to about the same degree as cystin, but after a few weeks the percentage of amino-acids excreted (in the form of sulphates) decreased, possibly in part from metabolic disturbance, partly from injury to the kidneys. Tests were made with rats to determine whether djenkol acid could replace cystin in the food, but the results were not conclusive. The eating of the beans by normal persons was followed by distinct increase in sulphur excreted, but the extent of this varies greatly. One man after eating two beans only suffered from anuria for two days; although he had taken only about 150 mgm. more sulphur he excreted 1,980 mgm. of oxidized sulphur and 2,980 mgm. total sulphur. Clearly, other sources of sulphur must have been mobilized. *H. H. S.*

GREVENSTUK (A.). Over Renghas- en Japanlak-vergiftiging.—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1935. Nov. 26. Vol. 75. No. 24. pp. 2065–2076. [19 refs.]

REVIEWS AND NOTICES.

WU LIEN-TEH [M.A., M.D., etc.], CHUN (J. W. H.) [M.B., B.C.], POLLITZER (R.) [M.D.] & WU (C. Y.) [M.B., B.S.]. **Plague : a Manual for Medical and Public Health Workers.**—pp. xxxiii+547. With 103 illustrations (6 coloured). 1936. Shanghai : Weishengshu National Quarantine Service, 2 Peking Road. [C.S. $10·00 ; 15s.]

A whole manual on one disease, of 547 pages with 103 illustrations, must betoken especial importance of the disease itself and a pressing need for instruction. This work is obviously intended for the English reader. The importance of plague as a devastating disease, which may attack any country, is worldwide, while the need for instruction is that of " medical and public health workers." It is stated in the preface that " the main aim of the authors has been to provide a practical guide to the prevention and treatment of plague." A perusal of the work shows that this aim has been fulfilled. The requirements of China and its special problems naturally occupy a prominent place in the manual and many of the illustrations have the same local significance, but any worker in any country will find some of the information he requires in this manual and will doubtless avail himself of it. There are 12 chapters in all, covering the subjects of the historical aspects of plague, its bacteriology, immunology, pathology, laboratory diagnosis, hosts and carriers, insect vectors, clinical features, therapy and personal prophylaxis, epidemiological factors, general prophylaxis and management of epidemics, and the problem of ship-borne plague. For each of these chapters one or the other of the authors is responsible. Even the medico-legal aspect of plague, as illustrated by the crime designated the " Plague Bacilli Case " is given, in an appendix, the notice due to a remarkable important event. It is certain that the sad tale of laboratory infections, as recorded here, would have to be extended if it were intended to be complete. Such cases are not always, or even commonly, published. Certain examples are, however, given in fair detail and serve the purpose of illustrating an ever-present danger for plague workers. A reference might perhaps have been made to the remarkable immunity of the staff of the laboratories engaged in plague routine or investigation, due, as one would infer, to their being regularly vaccinated. The sensitive guineapig test has shown, moreover, in some instances that the laboratories did not lack the presence of infected fleas. This seems to be a strong argument for the efficacy of the usual method of vaccination.

In view of the importance which is now attached to selvatic or wild rodent plague it is interesting to turn to the observations on this subject which are scattered through the chapter " Hosts and Carriers." The chapter makes reference at the outset to the " slogan," " No rats, no plague." It is therefore reassuring to find that the author of this chapter, Dr. Wu Lien Teh himself, maintains " that generally speaking the danger of a spread of plague from wild to domestic rodents is more apparent than real."

As Manchuria is classed, with other localities, as one of the principal foci of pneumonic plague, it was only to be expected that that form of disease would receive special treatment in this manual. In finding an answer to the question *how* pneumonic epidemics arise, the authors examine into the possibility of human pneumonic plague arising *de novo*

from rodents, but reject the idea in order to reassert their well-known and well-reasoned opinion that pneumonic plague in its principal foci, "like Egypt, Madagascar, South East Russia, Transbaikalia and Manchuria . . . usually originated in human cases with secondary lung involvement." On the further question of *why* pneumonic epidemics arise, it is excusable to find a greater dubiety, inasmuch as controversy is still as acute as ever on the reason for development of the epidemic or pandemic characters of any disease.

The illustrations throughout the work are clearly reproduced and the cost of it is very moderate. It should undoubtedly become a source of reference to many others than those for whose use it has been primarily designed. *W. F. Harvey.*

HUARD (P.) & MEYER-MAY (J.). **Les abcès du foie.** Préface du Professeur A. GOSSET. [Liver Abscesses.]—pp. viii+390. With 98 figs. 1936. Paris: Masson et Cie., 120 Boulevard Saint-Germain. [65 francs.]

This is a very detailed account of liver abscess based on 150 cases seen or collected by the authors in Tonking, where they are of the opinion the disease is rarely caused by amoebae. The account therefore promised to be of great interest and importance in differentiating the large proportion of their cases that they regarded as being of unknown aetiology, for the pus of 60 out of 93 cases examined at the time (they were treated by the open operation) was free from both amoebae and bacteria, in 7 it contained amoebae and in 27 several varieties of cocci and four species of bacteria were found, not more than 4 being due to any one organism.

After summarizing in tables their own data and those of other workers, including experience in India, they describe the anatomy and blood vessels of the organ and the aetiology just mentioned. A detailed account of the pathological anatomy and complications, illustrated with diagrams of the spread of the infection to neighbouring organs and cavities, unexpectedly reveals precisely the picture of amoebic liver abscess of other warm countries, and the clinical picture also shows nothing indicative of a new and previously undescribed form of abscess in the liver. The differential diagnosis is well described, with illustrative cases somewhat condensed from the fuller accounts of all of them at the end of the volume. A noteworthy feature of this section is exploratory puncture, on the lines of spleen puncture, to allow of microscopical examinations of the material thus obtained; by this means cancer, tuberculosis and other conditions were recognized in some suspected liver abscess cases. Still more interesting is the section on X-ray examinations, for this includes the previous injection of lipiodol to allow the shape of an abscess cavity, and its communication with secondary abscesses or a bronchus, to be defined.

Turning to the section on treatment we find more of interest. First they glance at that in use in other countries, and come to the remarkable conclusion that the difference in the methods used in some can only be explained by a difference in the class of abscesses met with. In discussing medical treatment they agree with others that arsenical, yatren, and bismuth preparations, etc., are all useless in congestive hepatitis, and only emetine is effective; but they differ in holding that the latter drug is equally curative of both amoebic and non-amoebic hepatitis. The now generally used aspiration and emetine treatment of liver

abscesses is rejected by the authors, although they hold that the previous use of emetine reduces considerably the incidence of abscesses, and they advocate courses of ten days at a time in the treatment of both acute and chronic hepatitis. A full description of the open operations which they always adopt is then given, including eleven incisions with extensions of some, aided by radiology, to enable the abscess to be reached by the most direct route. Their case mortality was 24 per cent. against 3·3 per cent. in K. K. Chatterji's 186 cases and nil in Ludlow's 47 cases (later 1 in 50 or 2 per cent.) by the aspiration method, which they quote. They also admit that in 74·3 per cent. of their 35 fatal cases the pus was sterile, and attribute the high mortality to "cachexia" and complications; but these are equally present in Calcutta cases. They deal with the rare secondary haemorrhages, and only mention secondary sepsis, which does not appear to have been by any means excluded as an important cause of death; as it used to be after the open operation in India.

We may now return to the main thesis of the authors, namely, that two-thirds (they say three-fourths in one place) of their liver abscess cases are of unknown aetiology and sterile in spite of their differing in no way from cases seen in other warm climates. Their assertion that only seven per cent. of their total cases were of amoebic origin appears to be based solely on the fact that that organism was only found in the pus of 7 out of 93 cases examined at the time of opening the abscess; but as emetine was given in a number of the non-amoebic cases in which the medical treatment is mentioned in their notes, and is advocated by them in all cases of hepatitis, such negative results in no way exclude an amoebic origin. Indeed they quote Constantini as saying that thanks to emetine, a liver abscess will no longer contain living amoebae. This appears to be a far more likely explanation of their results than that two-thirds of Tonking cases are due to some other distinct unknown cause, yet produce precisely the same pathological changes and clinical course as amoebic cases seen elsewhere. If that is so their advocacy of the open operation, with eight to twelve times the mortality following aspiration in Korea (Ludlow) and in Calcutta, falls to the ground, in spite of the advanced surgical technique displayed in their work. The monograph is worthy of careful study.

Leonard Rogers.

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